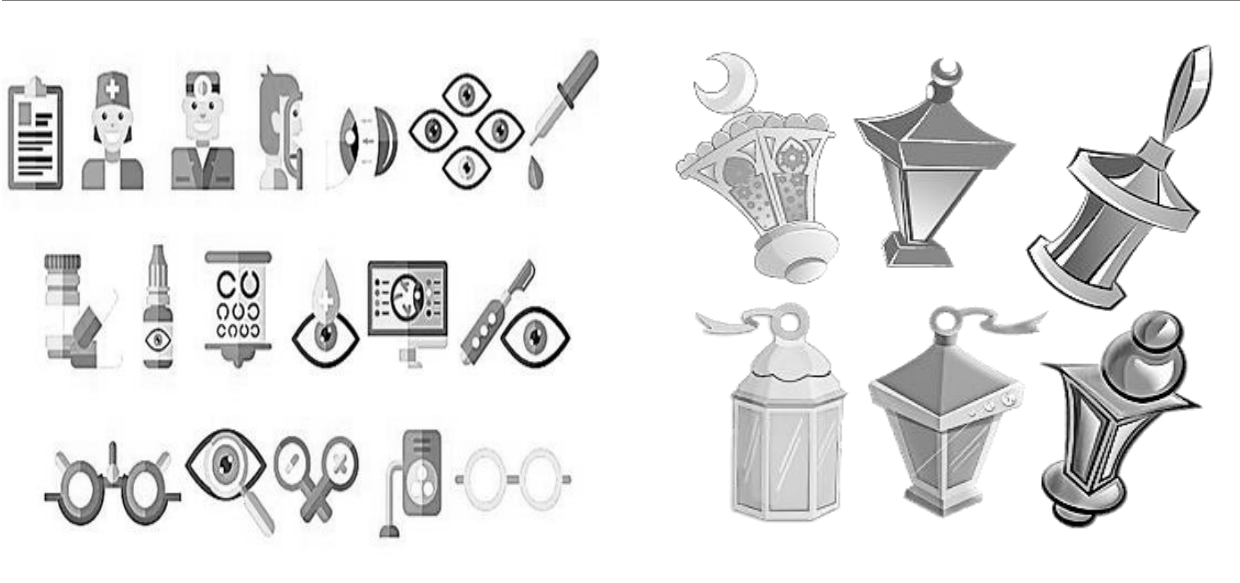


Final Revision Part (1)

Happy Ramadan with



Happy ophthalmology

Dr. AbdulRahman Al-Hosary

Uveal tract pI

Cornea p4

Sclera p7

Orbit p8

lacrimal system pii

Conjunctiva p13

Eyelids p19

Ocular trauma p25

Happy ophthalmology series:

 **Happy Final revision (2 parts):**  

 Smart view of the whole curriculum in tables.

 **Happy Exams with model answers:**

 **How to answer?**  **Exams of previous years with answers (as whole exams and also arranged on chapters).**

 **Happy MCQs:**

 Official MCQs arranged , organized and revised.

 Examples from exams of previous years with answers.

Happy clinical and oral:

 **Clinical tests.**

 **Clinical decision making.**

 **Oral notes.**

"من سلك طريقا يلتمس فيه علما، سهل الله له طريقا إلى الجنة.. وإن الملائكة لتضع أجنحتها لطالب العلم رضا بما يصنع.. وإن العالم ليستغفر له من في السماوات ومن في الأرض حتى الحيتان في الماء.. وإن فضل العالم على العابد كفضل القمر ليلة البدر على سائر الكواكب.. وإن الأنبياء لم يورثوا دينارا ولا درهما وإنما ورثوا العلم.. فمن أخذه أخذ بحظ وافر"

صدق رسول الله -صلى الله عليه وسلم-

VISIT...

LANZAROTE
Caliente.COM

	Uveal tract	
	Acute Irido-cyclitis	Chronic Irido-cyclitis
Definition	- Acute inflammation of the iris and the ciliary body (anterior part of uveal tract). - Both are usually inflamed together as they share a common blood supply.	Extremely chronic inflammation of the iris and the ciliary body which has gradual progressive course with exacerbations and remissions.
Etiology	<u>Exogenous</u>: During Trauma or Surgery Through Mechanical injury or Invasion of micro-organisms <u>Endogenous</u>: 2^{ty} to another diseases either: - Local e.g. Keratitis, long-standing RD, Retinoblastoma, MM & lens dislocation. - Systemic e.g. Arthritis, Bacterial (esp. chronic), viral, fungal and parasitic infections.	
Symptoms	- Ocular pain (neuralgic pain due to irritation of the 5th nerve endings and spasm of the ciliary muscle – ↑ed at night – referred to eyebrows). - Headache (in the forehead & around the eye) - Photophobia, Blepharo-spasm and Lacrimation. ↓ed vision (due to affection of the different ocular tissues – see signs).	- Ocular pain "milder". ↓ed vision: "gradual but more marked with prolonged time".
Signs	<u>Eyelids</u>: edema. <u>Conjunctiva</u>: ciliary injection <u>Cornea</u>: 1. Edema. 2. KPs (inflammatory cells on the back of the corneal endothelium detected by slit lamp – pathognomonic sign). <u>Anterior chamber</u>: 1. Aqueous inflammatory cells 2. Aqueous flare (due to leakage of plasma proteins – earliest sign). 3. Hypopyon (sterile pus). 4. Hyphema. 5. ↓ed IOP (due to inflamed CB) or ↑ed IOP (due to plasmoid aqueous). <u>Iris</u>: muddy with lost pattern. <u>Pupil</u>: miosis (due to straightening of vessels and spasm of the sphincter muscles) <u>Ciliary Body</u>: tenderness <u>Lens</u>: pigments precipitates <u>Vitreous</u>: inflammatory cells.	<u>Conjunctiva</u>: ciliary injection. <u>Cornea</u>: KPs "large mutton fat or small". <u>Anterior chamber</u>: deep or funnel. <u>N.B.</u> acute or sub-acute ↑ed IOP may occur periodically (Hypertensive irido-cyclitic crisis of Posner and Schlossman)- associated with flare and more KPs. <u>Iris</u>: muddy with atrophic patches. <u>Ciliary Body</u>: tenderness. <u>Vitreous</u>: dust-like opacities.
Complications	<u>Synechiae formation</u>: 1. <u>Peripheral anterior synechiae</u>: peripheral adhesions between the iris and the cornea. 2. <u>Posterior synechiae</u>: adhesions between the iris and the lens – may be: - Mild synechiae: adhesion at one or few points manifesting itself with pupil dilatation as a festooned pupil - Severe ring synechiae: adhesions occlude the pupil ⇒ iris bombe. - Severe total synechiae: adhesions plastering the iris to the lens. <u>Secondary glaucoma</u>: due to plasmoid aqueous and synechiae formation <u>Complicated cataract</u>: may be due to prolonged treatment with steroids. <u>Cystoid macular edema</u>: due to the effect of inflammatory mediators on the retinal vessels. <u>Cyclitic membrane</u>: 1. Site: behind the lens. 2. Cause: organization of exudates and vascularization from the ciliary body. 3. Fate: contraction of the membrane ⇒ CB detachment ⇒ ↓ed aqueous ⇒ Hypotony ⇒ Atrophia bulbi (small soft blind painful organized eye) and <u>Tractional RD</u>.	
		<u>On the iris</u>: 1. Iris atrophy 2. Rubeosis iridis ⇒ neo-vascular glaucoma. 3. <u>Iris nodules</u>: In TB: Koeppe nodules. in syphilis: Busacca nodules. <u>On the cornea</u>: 1. Band shaped keratopathy (horizontal strip of degeneration & Ca²⁺ deposition) 2. Sclerosing kerato-uveitis (infiltrations involve the 3 structures) <u>On the whole eye</u>: 1. Phthisis bulbi (small soft blind painful disorganized eye).

Investigations	🔪 <u>Laboratory</u>: ☁ For arthritis: HLA testing, RF and ANAs. ☁ For infections: CBC, ESR, tuberculin test, Wassermann test, VDRL and Complement fixation test. 🔪 <u>Radiological</u>: 📖 X-ray (chest., hand, feet and nasal sinuses), CT scan, MRI and US (to evaluate posterior segment of the eye). 🔪 <u>Tissue sampling</u>: 📖 conjunctival biopsy, AC aspiration and Vitreous aspiration (for cytology and PCR).
Treatment of Both Acute and Chronic Irido-cyclitis: 🔪 <u>Treatment of the Cause</u>: e.g. 1. for arthritis: steroids and immune-suppressive drugs. 2. for infections: antibiotics. 🔪 <u>Treatment of the Condition</u>: 📖 📖 📖 1. <u>Atropine</u>: the main line 📖 (N.B. Route and mechanism are the most important). ➤ Route: local ointment and eye drops. ➤ Mechanism: ↓ 📖 ☁ Spasm of ciliary muscle (cycloplegic). ⇒ ↓ Pain & Headache. ☁ Miosis (Mydriatic). ☁ Exudation from iris vessels and Posterior synechiae. ➤ Toxicity: 📖 📖 ☁ Caused by systemic absorption due to over dose. ☁ Manifested as fever, flushing, fits, tachycardia, dry skin and mouth. ☁ Prevented by proper dose and ointment use (instead of drops) in children. ☁ Treated by stopping atropine, giving pilocarpine (anti-dote) and cold compresses. ➤ Sensitivity: 📖 📖 ☁ Caused by allergic reaction with normal dose in any age or form ☁ Manifested as allergic dermatitis and follicular conjunctivitis. ☁ Treated by stopping Atropine, giving Cyclopentolate (substitute) and local steroids. ➤ Dangers (of mydriatics): ☁ Induce dry eye with prolonged use. ☁ Induce angle closure glaucoma in patients with narrow angle. ☁ Prevent pupil monitoring in patients with concussion and during anesthesia. ☁ Cyclopentolate (Atropine substitute) may induce psychic disturbances esp. in kids. 2. <u>Steroids</u>: 📖 📖 (N.B. Route and mechanism are the most important). ➤ Route: local ointment and eye drops and Systemic oral medication (both should tapered gradually). ➤ Mechanism: ↓ 📖 ☁ Inflammation. ☁ Allergy. ☁ Posterior synechiae. ➤ Dangers (of local steroids): ☁ Induce Glaucoma and Cataract. ☁ ↑ Susceptibility to infections ☁ Reactivate dormant infections e.g. herpes. ☁ Delay wound healing e.g. in CU. ➤ Dangers (of systemic steroids): ☁ DM, Hypertension and Peptic ulcer. ☁ Muscle wasting, osteoporosis. and Psychic disturbances. ☁ Cataract and Glaucoma. ☁ Reactivation of dormant infections e.g. TB , Cushingoid state Or Acute adrenal insufficiency with sudden withdrawal.	3. <u>Non-steroidal anti-inflammatory drugs (NSAIDs)</u>: 📖 📖 ➤ Route: Systemic Diclofenac or Ketorolac. ➤ Mechanism: inhibit prostaglandin synthesis ⇒ ↓ Inflammation. ➤ Indications: given in combination or as a substitute of steroids if contraindicated. 4. <u>Analgesics</u>: e.g. salicylates (systemic). 5. <u>Hot fomentations</u>: ↓ pain and improve the circulation (local effect). 6. <u>Corneal protectors</u>: e.g. dark glasses to ↓ photophobia (local effect). 🔪 <u>Treatment of the Complications</u>: 1. <u>Secondary glaucoma</u>: ➤ In the acute stage: treated medically as open angle glaucoma e.g. by Carbonic anhydrase inhibitors & Beta-blockers. (don't stop atropine). ➤ In the chronic stage: treated surgically as closed angle glaucoma e.g. by Peripheral iridectomy or trabeculectomy. 2. <u>Complicated cataract</u>: guarded removal of the cataract (after subsidence of signs of irido-cyclitis for at least 6 months). 3. <u>Cyclitic membrane</u>: removed by pars plana vitrectomy (provided that light projection is good). 4. <u>Blind painful eye (Atrophia or Phthisis bulbi)</u>: enucleation (excision of the whole eyeball) or retro-bulbar injection of alcohol.

Non-suppurative choroiditis		Signs: 👁️ Yellowish areas: 👁️👁️ ➤ Number, Shape & Site: one or more with ill margins deep to retinal vessels. ➤ Detected by: fundus examination. ➤ Fate: become white with pigmented margins in the healing stage. 👁️ Black spots: floating in the vitreous.
Definition: Non suppurative inflammation of the choroid (granulomatous or exudative).		
Symptoms: 👁️ Diminution of vision: due to retinal lesions and vitreous opacities. 👁️ Photopsia (flashes of light): due to retinal irritation. 👁️ Metamorphopsia: due to retinal edema. 👁️ Micropsia or Macropsia. 👁️ Scotoma.		
Treatment: the same as Irido-cylcitis (but steroids are the main line i.e. more important than Atropine).		
	Endophthalmitis	Panophthalmitis
Definition	Purulent (suppurative) inflammation <u>including</u> the choroid and intraocular tissues.	
	not <u>including</u> the outer coat of the eye (cornea and sclera) and Tenon's capsule.	<u>including</u> the outer coat of the eye, Tenon's capsule and the orbit.
Etiology	👁️ <u>Exogenous:</u> During: Trauma or Surgery. Through: Mechanical, Physical, Chemical injury or Infections . 👁️ <u>Endogenous:</u> 2 ^{ry} to Infections , Vasculitis or Neoplasms. 💀 <u>N.B.</u> 1 Infections (either endogenous or exogenous) are the most common cause.	
	💀 <u>N.B.</u> 2 Infections are mainly bacterial or fungal.	
Symptoms	☁️ <u>General symptoms:</u> Fever, Anorexia, Headache and Malaise.	
	☁️ <u>Ocular pain</u> neuralgic – dull aching): severe.	☁️ <u>Ocular pain:</u> very severe.
	☁️ <u>↓ed vision:</u> down to PL.	☁️ <u>lost vision:</u> no PL.
Signs	☁️ <u>Eyelids:</u> edema.	☁️ <u>Eyelids:</u> severe edema.
	☁️ <u>Conjunctiva:</u> ciliary injection.	☁️ <u>Conjunctiva:</u> ciliary injection.
	☁️ <u>Cornea:</u> edema .	☁️ <u>Cornea:</u> abscess.
	☁️ <u>AC:</u> inflammatory cells and pus (hypopyon).	☁️ <u>AC:</u> inflammatory cells and pus (hypopyon).
	☁️ <u>Vitreous:</u> inflammatory cells and pus (yellow reflex).	☁️ <u>Vitreous:</u> inflammatory cells and pus (yellow reflex).
		☁️ <u>Proptosis and Limited eye movements.</u>
		☁️ <u>Spread:</u> to cavernous sinus.
Manage- ment	👁️ <u>Hospitalization & Evaluation:</u> by AC and Vitreous aspiration for smears (Gram and Giemsa), culture & sensitivity test	
	👁️ <u>Treatment of cause:</u> 1. <u>For bacterial infections:</u> extensive Anti-biotics by the following routes and doses 🙌🙌 ➤ Intravitreal: ☁️ Vancomycin (1 mg) to cover gram +ve organisms. ☁️ Ceftazidime (2.25 mg) Or Amikacin (0.4 mg) to cover gram -ve organisms. ➤ Sub-conjunctival: ☁️ Vancomycin (25 mg). ☁️ Ceftazidime (100 mg) Or Amikacin (20 mg). ➤ Topical: ☁️ Vancomycin drops (50 mg/ml). ☁️ Ceftazidime drops (50 mg/ml) Or fortified Gentamycin drops (15 mg/ml). ➤ Systemic: ☁️ Ciprofloxacin IV or IM every 12 hours for 5 days (500 mg). ☁️ Amikacin IV or IM every 8 hours for 5 days (14 mg/Kg) . 💀 <u>N.B.</u> Monitoring kidney functions is important for fear of nephrotoxicity of ABs.	
	2. <u>For fungal infections:</u> extensive Anti-fungal (Amphotericin B) by the following routes 🙌🙌 ➤ Intravitreal. ➤ Topical ➤ Systemic.	
	👁️ <u>Treatment of condition:</u> 🙌	👁️ <u>Treatment of condition:</u> 🙌
	1. <u>Steroids:</u> ➤ Route: topical and /or systemic ➤ Timing: at least 24 hours after starting intensive antibiotic therapy.	1. <u>Evisceration:</u> ➤ Definition: evacuation of all contents of the eyeball. ➤ Indication: blind painful cases to stop pain and avoid spread of infection.
	2. <u>Vitrectomy:</u> in severe cases not responding to other measures.	
	💀 <u>N.B.</u> Enucleation "excision of the whole eyeball" is contraindicated in to avoid spread of infection to the meninges along optic nerve which is cut in this operation.	

	Cornea		
	Bacterial (Hypopyon) ulcer	Herpes simplex Keratitis	Herpes zoster ophthalmicus
Definition	Inflammation of the cornea in which there is destruction of the epithelium (and also the underlying stroma in many cases) of an area of the cornea due to		
Etiology	<u>Predisposing factors: e.g.</u> ➤ Traumatic abrasion as in rubbing lashes. ➤ Corneal anesthesia. ➤ Chronic conjunctivitis. ➤ Chronic dacryocystitis. ➤ Exposure keratopathy. <u>Causative organisms: bacteria e.g.</u> ➤ Pneumococci "main organism". ➤ Invasive organisms (don't need PDF) e.g. Listeria N. gonorrhea, C. diphtheria, and H. aegypticus.	<u>Predisposing factors: activating the virus e.g.</u> ➤ Immunosuppressant drugs e.g. steroids. ➤ Fever. ➤ Stress. <u>Causative organisms:</u> ➤ Herpes Simplex virus.	<u>Predisposing factors: e.g.</u> ➤ Old age and Immuno-compromised conditions. <u>Causative organisms:</u> ➤ Herpes zoster virus.
Symptoms	(General rules) ➤ Ocular pain: "neuralgic". ➤ Photophobia , Blepharo-spasm and Lacrimation. ➤ ↓ed vision. (⚠N.B. In HSK There's corneal hypoesthesia).		
Signs	<u>Eyelids and Conjunctiva:</u> edema. <u>Cornea:</u> 1. Acute serpiginous CU (Hypopyon CU). ➤ Central with 2 edges (central edge ⇒ undermined advancing and peripheral edge ⇒ sloping healing). ➤ There is infiltration in the bed and the edges of the epithelial defect. ➤ Has tendency to perforate. 2. Staining: fluorescein 1% ⇒ green color in blue light 3. Loss of corneal luster. <u>Anterior chamber:</u> cells, flare and Hypopyon.	<u>Eyelids:</u> vesicles. <u>Conjunctiva:</u> follicular conjunctivitis. <u>Cornea:</u> 1. CU description: ➤ Epithelial infiltration: punctuate ⇒ striate "linear" ⇒ stellate ⇒ dendritic "branching ending in round knobs" ⇒ amoeboid ⇒ geographic. ➤ Ulcer bed: insensitive. ➤ Superficial non-vascularized CU. 2. Staining: ➤ Ulcer bed: fluorescein. ➤ Diseased epithelialized margins: Rose Bengal.	<u>Corneal lesions:</u> 1. Punctate epithelial keratitis. 2. Micro- dendrites. 3. Nummular keratitis. 4. Disciform keratitis. <u>Other ocular lesions:</u> 1. Muco-purulent conjunctivitis. 2. Anterior uveitis 3. Scleritis and Epis-scleritis 4. Acute retinal necrosis. <u>Systemic lesions:</u> 1. Prodroma: Fever, malaise, headache, neuralgia along distribution of nerves. 2. Skin lesions: papules ⇒ pustules and crusting ulcers ⇒ healing by punched-out scars (unilateral along 1 or more of 3 branches of the ophthalmic nerve). 3. Neurological manifestations: cranial nerve (2,3,4,5) affection, encephalitis and post herpetic neuralgia.
Complications of any CU (General rules)	➤ Anterior uveitis ⇒ ➤ Synechia formation, etc.... ➤ Secondary glaucoma. ➤ Corneal opacity: nebula, macula or leucoma due to BM and stroma destruction ➤ Keratectasia: bulging of thin weak corneal scar "cicatrix". ➤ Descematocele: protrusion of Descemet's membrane. ➤ Actual perforation: which may be complicated by: 1. If the perforation is large: ➤ Subluxation and dislocation of the lens: due to sudden lowering of the IOP. ➤ Intraocular hemorrhage. ➤ Endophthalmitis: the most serious complication. 2. If the perforation is small: ➤ Corneal fistula: in which epithelium migrates inwards to line the CU ⇒ track. ➤ Epithelization: the epithelium migrate furthermore to line the back of the cornea, the anterior surface of the iris and the angle of the anterior chamber. ➤ Peripheral Anterior Synechiae: as the anterior chamber becomes flat and the cornea becomes adherent to the iris (first adhesions are fibrinous then fibrous). ➤ Secondary glaucoma: due to synechiae formation and angle epithelization. ➤ Leucoma adherent. ➤ Anterior staphyloma bulging of weak corneal scar lined by atrophic iris tissue. ➤ Complicated cataract.		

Invest.	✎ <u>Corneal scraping with culture and sensitivity test:</u> to confirm the etiologic diagnosis before starting the specific treatment of the cause (<i>write causative organisms</i>)		
Treatment	✎ <u>Prophylaxis:</u> by Using protective measures in patients with high risk and Early treatment of diseases predisposing to some corneal ulcers.		
	✎ <u>Treatment of Cause:</u> Topical broad spectrum ABs e.g. 1. Fluoroquinolones: e.g. Ciprofloxacin 0.3% "covers organisms except some staph aureus and pseudomonas" 2. Combination of "fortified" Aminoglycosides and Cephalosporines.	✎ <u>Treatment of Cause:</u> Antiviral drugs e.g. 1. Acyclovir 3% ointment 5 times / day. 2. Adenosine- arabinoside 3% ointment 5 times / day 3. Trifluoro-thymidine 1% eye drops 5 times / day. ⚠ N.B. All act by interfering with viral replication.	✎ <u>Treatment of Cause:</u> Acyclovir by following routes: 1. Topical. ✎ <u>Systemic</u> 800mg tablets (5 times daily for 7days). ✎ <u>Skin creams.</u> (and also steroid- antibiotic skin cream)
	✎ <u>Treatment of Condition:</u> ➤ Atropine sulphate ➤ Hot fomentations. ➤ Corneal protectors e.g. patching and dark glasses. (<i>write all of them as irido-cyclitis</i>). ➤ Vitamins A, B and C (supportive). ☁ Corticosteroids (contraindicated especially in herpetic ulcers as they delay wound healing and may cause perforation, However, short-term could be used only in recently healed ulcers to ↓ corneal scarring and therefore, affection on visual acuity). ✎ <u>Treatment of Complications:</u> ☁ <u>Debridement followed by intensive topical medications:</u> to remove the infected epithelium ☁ <u>keratoplasty (either lamellar or penetrating):</u> to manage opacified cornea. ☁ <u>Tarsorrhaphy and conjunctival flaps:</u> to promote healing in cases of exposure and loss of corneal sensation. ☁ <u>Paracentesis:</u> in cases of descemetocoele (impending perforation) and in cases of Hypopyon with secondary glaucoma ☁ <u>Tissue adhesive glue:</u> for small perforation. ☁ <u>Therapeutic corneal grafting:</u> for larger perforation. ☁ <u>Cautery:</u> by tincture iodine 7.5% or absolute alcohol.		
	Fungal CU	Protozoal CU	CU with lagophthalmos (Exposure keratopathy)
Etiology	✎ <u>Predisposing factors:</u> ➤ Foreign body touch (plant origin). ✎ <u>Causative organisms:</u> ➤ Fungal infections.	✎ <u>Predisposing factors:</u> ➤ Contact lens use with bad hygiene. ✎ <u>Causative organisms:</u> ➤ Protozoal infections.	Etiology: ✎ <u>Cause:</u> Lagophthalmos due to OO paralysis (usually due to facial n. paralysis). ✎ <u>Pathogenesis:</u> Lagophthalmos ⇒ dryness of the cornea + lack of corneal protection from minor trauma ⇒ necrosis and sloughing of superficial layers.
Symptoms	✎ The same as general rules (but the pain is severe with protozoal infections).		
Signs	✎ Atypical CU. (not easy to diagnose).		
Comp.	✎ The same as general rules.		
Invest.	✎ The same as general rules.		
Treatment	✎ <u>Prophylaxis, Treatment of the condition and complications:</u> as general rules.		
	✎ <u>Treatment of the cause:</u> ➤ Antifungal e.g. Amphotericium B	✎ <u>Treatment of the cause:</u> ➤ Anti-acanthamebic (protozoal).	✎ <u>Treatment of the cause:</u> ☁ <u>lagophthalmos:</u> by lateral tarsorrhaphy ☁ <u>Facial nerve paralysis.</u> ✎ <u>Treatment of the condition and complications:</u> as general rules.
Corneal anesthesia (neuropathic keratopathy)			Clinical picture and Complications: as general rules but sensations are lost.
Etiology: ✎ <u>Cause:</u> Loss of corneal sensation due to destruction of the trigeminal ganglion due to: ☁ Trauma e.g. in fracture base of the skull. ☁ Inflammation e.g. in gummatous meningitis. ☁ Iatrogenic e.g. after treatment of trigeminal neuralgia. ☁ Post HZO and herpes HSK. ✎ <u>Pathogenesis:</u> Corneal anesthesia ⇒ loss of corneal protection, loss of reflex blinking and lacrimation & loss of trophic nerve impulses.			Treatment: ✎ <u>Prophylaxis:</u> by: using protective measures waiting for recovery of sensations: ➤ Therapeutic soft contact lens. ➤ Temporary tarsorrhaphy. ✎ <u>Treatment of the cause:</u> ✎ <u>Treatment of the condition and complications:</u> the same as general rules.

Nutritional deficiency CU (keratomalacia)		Photophthalmia (snow blindness)																									
Etiology: ✎ Vitamin A deficiency caused by advanced starvation and marasmus in young children		Definition and Etiology: ✎ Superficial inflammation of cornea and conjunctiva <u>caused by</u> exposure to UV rays (usually following welding or skiing).																									
Clinical picture and Complications: as general rules but <i>specific features are:</i> ✎ Bilateral melting of the cornea. ✎ Minimal inflammatory response. ✎ Prolapse of the ocular contents when perforation occurs. ✎ 2 ^{ry} bacterial infection and Panophthalmitis. ✎ Blindness		Clinical picture and Complications: ✎ The same as general rules of CU + general rules of conjunctivitis <i>but:</i> ✎ Symptoms appear 4-6 hours after exposure "latent period".																									
Treatment: ✎ Treatment of the cause: <i>main line – by:</i> ➤ Large doses of Vitamin A: Systemic (5000 IU/day) and Local eye ointment. ➤ Improving the general condition. ✎ Treatment of the condition: the same as general rules. ✎ Treatment of the complications: the same as general rules.		Treatment: ✎ Prophylaxis: <i>by:</i> wearing dark glasses and protective goggles during exposure. ✎ Treatment of the condition: <i>by:</i> ➤ Frequent topical lubricants e.g. Methyl cellulose. ➤ Cold compresses. ➤ Vasoconstrictor eye drops e.g. Zinc sulfate. ➤ Bandage of both eyes for 1 day (till the epithelium heals).																									
Keratoconus		Corneal Vascularization																									
Definition: ✎ Apical protrusion of the cornea forming a cone- shaped deformity due to progressive central thinning of the stroma.																											
Incidence ✎ Bilateral in 85% of cases. ✎ Starts around puberty (10-20 years) and progresses for few years.	Associations: ✎ Ocular diseases e.g. spring catarrh. ✎ Systemic diseases e.g. Down syndrome and Marfan syndrome.																										
Symptoms: ✎ Frequent changing of glasses due to rapidly progressive myopia and astigmatism.																											
Signs: ✎ ✎ ✎ Apical protrusion of the cornea forming a cone- shaped deformity. ✎ Corneal thinning, scarring and opacities. ✎ <u>Fleischer ring:</u> iron deposits at the base of the cone. ✎ <u>Munson's sign:</u> angulations of the lower lid on downward gaze.																											
Complications: ✎ Rapidly progressive myopia and astigmatism.																											
Investigations: ✎ Corneal topography: helps in early diagnosis.																											
Treatment: ✎ Treatment of the condition: <i>new techniques to improve or stabilize the condition e.g.</i> ➤ Intra corneal segments and Cross linking. ✎ Treatment of the complications: <i>the same as astigmatism treatment i.e.</i> ➤ Spectacles, Rigid CL and Penetrating keratoplasty (PKP) – discuss details.																											
		<table><tr><td></td><th>Superficial</th><th>Deep</th></tr><tr><td>Origin</td><td>Conjunctival vessels</td><td>Anterior ciliary vessels</td></tr><tr><td>Site</td><td>Vessels run in the superficial stroma</td><td>Vessels run in deep 2/3 stroma</td></tr><tr><td>Surface</td><td>Irregular (raised by blood vessels)</td><td>Smooth.</td></tr><tr><td>Shape</td><td>Vessels branch dichotomously.</td><td>Vessels run parallel.</td></tr><tr><td>Color</td><td>Bright red well defined.</td><td>Dark red ill-defined.</td></tr><tr><td>Extent</td><td>Vessels cross the limbus.</td><td>Vessels end abruptly at the limbus.</td></tr><tr><td>Causes</td><td>✎ Superficial CU. ✎ Pannus. ✎ Pterygium. ✎ Trichiasis ✎ Ariboflavinosis.</td><td>✎ Deep CU. ✎ Interstitial keratitis.</td></tr></table>			Superficial	Deep	Origin	Conjunctival vessels	Anterior ciliary vessels	Site	Vessels run in the superficial stroma	Vessels run in deep 2/3 stroma	Surface	Irregular (raised by blood vessels)	Smooth.	Shape	Vessels branch dichotomously.	Vessels run parallel.	Color	Bright red well defined.	Dark red ill-defined.	Extent	Vessels cross the limbus.	Vessels end abruptly at the limbus.	Causes	✎ Superficial CU. ✎ Pannus. ✎ Pterygium. ✎ Trichiasis ✎ Ariboflavinosis.	✎ Deep CU. ✎ Interstitial keratitis.
	Superficial	Deep																									
Origin	Conjunctival vessels	Anterior ciliary vessels																									
Site	Vessels run in the superficial stroma	Vessels run in deep 2/3 stroma																									
Surface	Irregular (raised by blood vessels)	Smooth.																									
Shape	Vessels branch dichotomously.	Vessels run parallel.																									
Color	Bright red well defined.	Dark red ill-defined.																									
Extent	Vessels cross the limbus.	Vessels end abruptly at the limbus.																									
Causes	✎ Superficial CU. ✎ Pannus. ✎ Pterygium. ✎ Trichiasis ✎ Ariboflavinosis.	✎ Deep CU. ✎ Interstitial keratitis.																									
		Corneal opacities																									
		Etiology and Incidence: ✎ Corneal opacities are common cause for blindness in Egypt that occur as a complication of different corneal lesions.																									
		Types: ✎ <u>According to density:</u> Nebula, Macula, Leucoma and Leucoma adherent. ✎ <u>According to site:</u> Peripheral, Paracentral, Central..																									
		Management: <i>depends on the site and the effect on visual acuity:</i> ✎ <u>Peripheral opacities with no effect on vision:</u> No treatment. ✎ <u>Peripheral opacities with some traction centrally (changing curvature):</u> Glasses or CL ✎ <u>Paracentral significant opacities with positive mydriatic test:</u> Topical mydriatic or Visual iridectomy. ✎ <u>Central significant opacities:</u> Keratoplasty.																									

	Sclera		
	Epi-Scleritis	Scleritis	Staphyloma
Definition	✎ Inflammation of the epi-scleral tissue. ✎ It's more common in females.	✎ Inflammation of the all layers of the sclera. ✎ It is more serious than epi-scleritis.	✎ bulging (ectasia) of weak scar in the outer coat of the eye (cornea or sclera) lined by atrophic middle coat (uveal tissue).
Etiology	✎ Idiopathic. ✎ Collagen disease e.g. rheumatoid arthritis. ✎ Allergy: to an endogenous toxin as a septic or tuberculous focus.		✎ Corneal (Anterior) staphyloma: ➤ Bulging of weak corneal scar lined by atrophic iris tissue. ➤ It may be partial or total.
Symptoms	✎ Tenderness on pressing the globe. ✎ Discomfort and Lacrimation.	✎ Severe pain. ✎ Photophobia and Lacrimation.	
Signs	✎ Nodular type: ⊙ Distribution: lentil-sized well circumscribed nodule ⊙ Color: purple in due to dilatation of deep vessels. ⊙ Tender and the conjunctiva moves freely over it. ✎ diffuse type. ⊙ Distribution: affecting one (more frequent) or more sectors. ⊙ Color: purple due to dilatation of epi-scleral vessels.	✎ Anterior type: with 5 subtypes: ⊙ Nodular: deep nodule fixed to the sclera. ⊙ Diffuse. ⊙ Annular. ⊙ Necrotizing: serious ⇒ perforation of the globe ⊙ Scleromalacia perforans: melting of the sclera in a quiet eye ✎ Posterior type: ⊙ causes severe pain and uveitis with exudative RD.	✎ Scleral staphyloma: has 4 sub-types: ⊙ Intercalary staphyloma: ➤ Bulging of 2-3 mm zone of sclera and limbus (which is weakened by Schlemm's canal) lined by atrophic iris and peripheral anterior synechiae. ⊙ Ciliary staphyloma: ➤ Bulging of weak scleral scar lined by atrophic ciliary body tissue. ⊙ Equatorial staphyloma: ➤ Bulging of weak scleral scar lined by atrophic chorio-retinal tissue. ⊙ Posterior staphyloma: ➤ Bulging of weak scleral scar Temporal to the optic disc lined by atrophic chorio-retinal tissue. ➤ It occurs in high degree axial myopia and seen by ophthalmoscope.
Complications		✎ Sclerosing keratitis: corneal opacity develops near the scleral nodule. ✎ Scleral staphyloma: due to weakness of the sclera ✎ Uveitis and exudative RD: with the posterior type.	
Treatment	✎ Treatment of Cause and Condition: ➤ Topical steroids (less effective in scleritis).	➤ Non-steroidal anti-inflammatory drugs (NSAIDs). ➤ Systemic steroids. ➤ Immunosuppressive drugs. ✎ Treatment of Complications.	

Orbit

Proptosis

Definition:

✎ Abnormal protrusion of the eyeball (Normally, a line between the middle of the upper and lower orbital margins just touches or misses the corneal apex through closed eyelids).

Etiology (Causes):

✎ Endocrinal (Dys-thyroid eye disease): commonest cause.

✎ Congenital: e.g. Dermoid cyst of the orbit and Meningocele.

✎ Traumatic: through inducing:

⊙ Retro-bulbar hematoma.

⊙ Surgical emphysema (air).

⊙ Arterio-Venous fistula "Carotid-Cavernous fistula".

✎ Inflammatory: may be:

⊙ Acute inflammation: e.g.

➤ Panophthalmitis.

➤ Orbital cellulitis.

➤ Cavernous sinus thrombosis.

➤ Orbital periostitis.

⊙ Chronic inflammation: may be:

➤ Non-specific e.g. orbital pseudo-tumor.

➤ Specific e.g. tuberculosis of the orbit.

✎ Neoplastic: may be:

⊙ Benign tumors: e.g.

➤ Hemangioma (commonest benign orbital tumor).

➤ Neuro-fibroma.

➤ Osteoma.

⊙ Malignant: tumors: may be:

➤ From orbital tissues e.g. Sarcomas..

➤ From orbital organs other than the eye e.g. lacrimal gland tumors

➤ From the eye (Intraocular tumors) e.g. retinoblastoma & MM of the choroid

➤ From orbital surroundings e.g. tumors of nose and sinuses.

➤ Metastatic and blood borne e.g. breast carcinoma, prostatic carcinoma,

neuroblastoma of suprarenal gland and leukemia (especially in children).

✎ Others: e.g.

⊙ Orbital varix.

⊙ Hydatid cyst.

⊙ Aneurysm and Blood cyst.

⊙ Bone disease e.g. Paget's disease.

Diagnosis:

📖 History taking:

1. Initially: mode of onset, course progress and duration of the complaint (e.g. acute conditions suggest inflammations and trauma, chronic slowly progressive conditions suggest malignancies and recurrent conditions suggest orbital varix, and pseudo-tumor).

2. Presence of associated symptoms: e.g. pain and diplopia (e.g. painful conditions suggest inflammations).

3. History of possible causes: e.g. trauma, nearby infection or systemic disease (e.g. thyrotoxicosis and malignancies).

📖 Examination:

1. Initially: exclude pseudo-proptosis which may be due to:

➤ Large globe e.g. in high myopia especially when unilateral.

➤ Buphthalmos (primary congenital glaucoma).

➤ Shallow orbit e.g. in craniofacial anomalies.

➤ Contra lateral enophthalmos.

2. General examination: looking for signs of systemic diseases (e.g. thyrotoxicosis and malignancies).

3. Local examination:

⊙ Inspection: looking for:

➤ Type of proptosis (unilateral or bilateral).

➤ Direction of proptosis (anterior, lateral, up or down).

➤ Associated local signs (e.g. signs of inflammation, edema and optic nerve affection).

⊙ Palpation: looking for:

➤ Orbital swelling (detect consistency and compressibility).

➤ Pulsations (e.g. in carotid cavernous fistula, vascular sarcoma or aneurysms).

⊙ Measurement of proptosis: done by:

➤ Simple ruler.

➤ Hertel's exophthalmometer (better).

📖 Investigations:

1. Laboratory:

⊙ For thyroid function: T₃, T₄ and thyroid stimulating hormone (TSH).

⊙ For infections:

➤ Generally: CBC (total and differential leucocytic count) and ESR.

➤ For TB: tuberculin test.

➤ For Syphilis: Wassermann test and VDRL.

➤ For Hydatid disease: Casoni test.

⊙ For leukemia: CBC (total and differential leucocytic count).

2. Radiological:

⊙ X-ray: enlarged optic foramen or orbital fissures, calcification, erosion, deviation, etc..

⊙ CT scan, MRI, US.

⊙ Carotid Angiography: for carotid cavernous fistula.

3. Ocular tissue sampling and biopsy:

⊙ Fine needle aspiration or excision biopsy is usually needed in orbital tumors

Endocrine Exophthalmos (Dys-thyroid ophthalmopathy – Thyroid eye disease)

Definition and Etiology:

✎ Changes in the eye and its accessory structures caused by thyroid gland dysfunction.

Clinical features:

1. Eye lid signs:

- ⊙ Upper lid retraction (Dalrymple sign): a white scleral rim is visible above the upper margin of the cornea in the primary position ⇒ staring look.
- ⊙ Upper lid lag (Von Greave sign): The upper lid does not follow the eyeball when looking downward.
- ⊙ Infrequent blinking (Stellwag sign).

2. Infiltrative Ophthalmopathy:

⊙ Pathogenesis:

- Proliferation of orbital fat with retention of fluids.
- Cellular infiltration by lymphocytes and plasma cells.
- Proliferation of Connective tissue.
- Accumulation of muco-polysaccharides.

⊙ Clinical signs:

- Injection, hyperemia, and chemosis of the conjunctiva.
- Edema of the eyelids.

3. Extraocular muscle affection:

⊙ Pathogenesis:

- Accumulation of muco-polysaccharides with retention of fluids.
- Subsequent degeneration and fibrosis of the muscle fibers.

⊙ Clinical signs:

➤ Weakness and limited ocular movements in various directions (up, out, down, in) ⇒ diplopia.

4. Compressive optic neuropathy: (similar to papillitis but ↓ of vision is slow)

⊙ Symptoms:

- Vision: slowly progressive impairment of central vision.
- V.F: central or para-central scotoma to red and green.

⊙ Signs:

- Pupil: Marcus-Gunn pupil (RAPD).
- V.F: central or para-central scotoma to red and green (with or without NFLD).
- Fundus examination: chorio-retinal folds and disc edema up to optic atrophy.

5. Exophthalmos: the commonest cause of both unilateral and bilateral proptosis – may be:

	Thyrotoxic Exophthalmos	Thyrotropic Exophthalmos
Cause	Increased thyroxin (Hyperthyroid state)	Increased TSH or EPS (Hypo or Eu-thyroid state)
Pathogenesis	Spasm of Muller's muscles	Increased orbital contents
Age	Adults	Middle-aged
Sex	Females more	Males more
Onset	Rapid	Insidious
Course	Benign	Malignant (more corneal damage)
Severity	Mild and Reducible	Severe and irreducible
Extra-ocular muscles	Normal	Affected (ophthalmoplegia)
Systemic manifestations	Present	Absent
BMR	Elevated	Normal

Complications:

✎ Corneal exposure keratopathy.

Treatment:

✎ Treatment of the cause: i.e. thyroid gland dysfunction.

✎ Treatment of the condition:

- Systemic steroids: in early cases with painful exophthalmos.
- Radiotherapy: when steroids are contraindicated or ineffective.
- Orbital decompression: in cases of severe proptosis (N.B. *Orbital decompression* means removal of one or more of the orbital walls).
- Extraocular muscle surgery: when there is diplopia in the primary or reading position
- ✎ Treatment of the complications: i.e. corneal affection.

Inflammatory Proptosis

Causes: see before.

Orbital pseudo-tumor:

✎ Definition: chronic non-specific inflammation involving the orbital tissues

✎ Etiology: Unknown but may be orbital immune reaction

✎ Treatment: ✎ Oral steroids (rapid and dramatic response within 2-3 days) – Radiotherapy - Immunosuppressive agents (in refractory cases).

Other causes: see later.

	Panophthalmitis	Orbital cellulites	CST	Deep orbital periostitis
Definition	🔪 Suppurative inflammation <u>including</u> the choroid, intraocular tissues, the outer coat, Tenon's capsule and orbit.	🔪 Acute suppurative inflammation of the orbital soft tissues	🔪 Thrombo-phlebitis of the cavernous sinus.	🔪 Acute suppurative inflammation of the deep orbital periosteum
Etiology	🔪 <u>Exogenous</u> : ➤ <u>During</u> : Trauma or Surgery. ➤ <u>Through</u> : Mechanical, Physical, Chemical injury or Infections	🔪 <u>Route of the infection</u> : spread from : ➤ Nasal sinuses (sinusitis). ➤ Blood borne		🔪 <u>Infective origin</u> : spreading from the neighboring parts.
	🔪 <u>Endogenous</u> : ➤ 2 ^{ly} to Infections , Vasculitis or Neoplasms. 💀 Infections are the commonest cause.		➤ The face. ➤ The orbit and the globe. ➤ The mouth and the pharynx. ➤ The middle ear and mastoid.	🔪 <u>non-infective injuries</u> .
	🔪 <u>Causative organisms</u> : ➤ Bacteria e.g. staphylococci, streptococci and pneumococci.			
	➤ Fungi (rare).			
Symptoms	🔪 <u>General symptoms</u> : Fever, Anorexia, Headache and Malaise (+ to ++)		🔪 <u>General symptoms</u> : Fever, Anorexia, Headache and Malaise (+++ to ++++)	
	🔪 <u>Local pain</u> neuralgic – dull aching): severe			
	🔪 <u>Vision</u> : early affected down to no PL.		🔪 <u>Vision</u> : early good (affected only late only if optic neuritis develops).	
Signs	☁ <u>Eyelids</u> : severe edema. ☁ <u>Conjunctiva</u> : ciliary injection – edema. ☁ Proptosis and Limited eye movements			
	☁ <u>Cornea</u> : abscess.	☁ Orbital abscess: points in the lower fornix or into the skin near the orbital margin.	☁ Papilledema and Dilated retinal veins.	☁ Proptosis is deviated to one side
	☁ <u>AC</u> : inflammatory cells and pus (hypopyon).		☁ Edema and tenderness of the mastoid region.	☁ Orbital cellulitis.
	☁ <u>Vitreous</u> : inflammatory cells and pus (yellow reflex).		☁ Papillitis.	☁ Superior orbital fissure syndrome.
	☁ <u>Spread</u> : to cavernous sinus.		☁ Paralysis of the lateral rectus (due to 6th nerve palsy).	☁ Sinus formation (e.g. in T.B).
Comp.	☁ Spread to cavernous sinus.	☁ Corneal exposure, hypoesthesia⇒ CU	☁ Death.	☁ Spread of infection to the brain ⇒ (meningitis and CST).
Management	🔪 <u>Hospitalization & Evaluation</u> : by AC & Vitreous aspiration.	🔪 <u>Prophylaxis and treatment of Cause</u> : Anti-biotics.		
	🔪 <u>Treatment of cause</u> : extensive Anti-biotics or Anti-fungal by different routes.	🔪 <u>Treatment of Condition</u> : ☁ Hot fomentation. ☁ If an abscess forms: incision and evacuation of pus.	🔪 <u>Treatment of Condition</u> : ☁ Anti-coagulants.	🔪 <u>Treatment of Condition</u> : ☁ exploratory orbitotomy. ☁ If an abscess forms: incision and evacuation of pus.
	🔪 <u>Treatment of condition</u> : Evisceration.	🔪 <u>Treatment of Condition</u> : e.g. CU		🔪 <u>Treatment of Condition</u> : e.g. CU
	"for details of treatment (see uveal 😊)"			
Enophthalmos			Operations of the orbit	
Definition:			🔪 <u>Orbitotomy</u> : removal of orbital tumors or doing orbital decompression in thyroid cases	
Retraction of the eyeball into the orbit (opposite to proptosis).			🔪 <u>Evisceration</u> : evacuation of the contents of the eyeball in endo- and pan-ophthalmitis.	
Causes:			🔪 <u>Enucleation</u> : excision of the eyeball in severe trauma, tumors and blind painful eye.	
🔪 Horner's syndrome: commonest cause. 🔪 Senile: due to absorption of orbital fat.			🔪 <u>Orbital exenteration</u> : removal of the orbital periosteum and all the orbital contents, lids and conjunctiva in malignant tumors of orbit or spreading to the orbit.	
🔪 Traumatic (why? ⇒ see trauma 😊) 🔪 Inflammatory. 🔪 Post-operative.				

lacrimal system

	Dry eye	Watery eye
Definition	dryness of cornea and conjunctiva due to ↓ed secretion of pre-corneal tear film mainly due to lacrimal gland dysfunction.	↑ed flow of tears onto the checks due to either: ☁ ↑ed secretion of tears (lacrimation). ☁ ↓ed drainage of tears (epiphora).
Etiology	✎ <u>Lacrimal gland dysfunction:</u> which may be: ➤ Congenital absence of the lacrimal gland. ➤ Inflammation of lacrimal gland (dacryoadenitis) e.g. in sarcoidosis. ➤ Tumors of lacrimal gland e.g. mixed lacrimal gland tumor. ➤ Kerato-conjunctivitis sicca: autoimmune disease ⇒ atrophy and fibrosis of the lacrimal gland, it occurs usually in females and may be associated with arthritis and dry mouth (<u>Sjogren's syndrome</u>). ✎ <u>Conjunctival scarring:</u> which may be due to: ➤ Trachoma. ➤ Chemical burns. ➤ Stevens-Johnson syndrome. ➤ Ocular cicatricial pemphigoid. ✎ <u>Meibomian gland dysfunction.</u>	☁ Lacrimation ✎ <u>Emotional conditions.</u> ✎ <u>Reflex lacrimation:</u> from ➤ Foreign body. ➤ Inflammation of the lid margin, conjunctiva, cornea, iris and ciliary body. ➤ Glaucoma, Errors of refraction, and Latent squint. ☁ Epiphora ✎ <u>Lacrimal pump failure:</u> due to abnormality in: ➤ Lid margin e.g. abnormality in the posterior border of the lid margin or ectropion. ➤ Orbicularis muscle e.g. Facial palsy. ✎ <u>Lacrimal pathway obstruction:</u> at the level of the puncti, canaliculi, lacrimal sac, naso-lacrimal duct or at the nose which may be: ➤ Congenital e.g. in punctual atresia & NLD obstruction. ➤ Inflammatory e.g. in trachoma, herpes & fungal infections. ➤ Traumatic e.g. in bony fractures, surgical trauma & foreign body as lashes. ➤ Tumors e.g. nasal polyps, maxillary tumors.
Clinical evaluation	✎ <u>The patient complains of:</u> irritation and foreign body sensation. ✎ <u>The doctor notes:</u> punctate epithelial erosion of the cornea. ✎ <u>The doctor apply 3 tests:</u> ☁ ➤ Rose Bengal staining: of degenerated epithelium of the conjunctiva and the cornea. ➤ Schirmer's test: to measures tear production by filter paper ⇒ ↓ed. ➤ Tear film Break-Up Time (BUT) ⇒ ↓ed.	✎ <u>Exclude causes of lacrimation.</u> ✎ <u>Regurgitation test:</u> reflux of pus or tears from the puncti in case of NLD obstruction. ✎ <u>Fluorescein dye test:</u> ➤ Instill a drop of fluorescein in the conjunctival sac and put a cotton pellet under the inferior turbinate of the nose and then irrigate with saline. ➤ If fluorescein isn't recovered & saline doesn't reach nose ⇒ complete block. ✎ <u>Dacryo-cystography:</u> Inject a radiocontrast medium and do X-ray at intervals to detect filling of the lacrimal system. ✎ <u>Plain X-ray:</u> For diagnosis of tumors or fractures.
Treatment	✎ <u>Treatment of the cause:</u> e.g. auto-immune cases by systemic steroids ✎ <u>Treatment of the condition:</u> by: ➤ Tear substitutes (e.g. eye drops and eye gel). ➤ Occlusion of the puncti (to reduce tear drainage). ➤ Protective glasses and contact lenses (to prevent corneal affection). ✎ <u>Treatment of the complications:</u> e.g. corneal erosions.	✎ <u>Treatment of the causes:</u> e.g. ➤ Ectropion and Facial palsy. ➤ Obstruction at the level of puncti or canaliculi: dilatation and probing. ➤ Congenital NLD obstruction: treated as congenital dacryocystitis (<i>write it</i>). ➤ Acquired NLD obstruction: treated as chronic dacryocystitis (<i>write it</i>).

	Congenital (Infantile) dacryocystitis	Chronic dacryocystitis	Acute dacryocystitis
Definition	✎ Inflammation of the lacrimal sac due to congenital imperforate Hasner's valve at the lower NLD end.	✎ Chronic inflammation of the lacrimal sac. ✎ It's the commonest lacrimal sac disorder	✎ Acute suppurative inflammation of the lacrimal sac.
Etiology	✎ NLD obstruction (Imperforate Hasner's valve).	✎ <u>Predisposing factors:</u> NLD obstruction. ✎ <u>Causative organisms:</u> ☼ Bacterial infections e.g. pneumococci, staphylococci and streptococci. ☼ Fungal infections.	✎ <u>Predisposing factors:</u> NLD obstruction. ✎ <u>Causative organisms:</u> ☼ Bacterial infections e.g. pneumococci, staphylococci and streptococci
Symptoms	✎ Epiphora (watery eye).	✎ Epiphora (watery eye). ✎ Discharge.	✎ Pain: severe. ✎ General symptoms of infections (FAHM).
Signs	✎ +ve regurgitation test: pressure over the lacrimal sac ⇒ reflux of clear fluid, mucus, muco-pus or sometimes frank pus.	✎ +ve regurgitation test: pressure over the swollen lacrimal sac ⇒ reflux of mucus or pus. ✎ Swelling of lacrimal sac: below the medial palpebral ligament. (plus clinical evaluation of watery eye).	✎ -ve regurgitation test: due to congestion of the epithelium of the canaliculi. ✎ Swelling of lacrimal sac: tender. ✎ Skin over the sac: red edematous. ✎ Abscess formation with fluctuation
Complications	✎ Recurrent conjunctivitis.	✎ <u>Ocular complications:</u> ☼ Chronic conjunctivitis. ☼ Hypopyon CU. ☼ Endophthalmitis. ✎ <u>Lacrimal complications:</u> ☼ Mucocele and pyocele. ☼ Lacrimal fistula. ☼ Epiphora, eczema and ectropion.	✎ <u>Orbital complications:</u> ☼ Orbital cellulitis. ☼ Cavernous sinus thrombosis. ✎ <u>Lacrimal complications:</u> ☼ Mucocele and pyocele: if canaliculi are obstructed. ☼ Lacrimal fistula: if the sac bursts anteriorly. ☼ Chronic dacryocystitis.
Treatment	✎ <u>Treatment of Condition:</u> ☼ <u>Conservative methods:</u> ➤ Include: ABs with sac hydrostatic massage. ➤ Aim: to remove any epithelial remnants. ➤ Duration: tried for a long period up to 6 months. ☼ <u>Probing:</u> successful if done carefully ☼ <u>Repeated syringing & irrigation with saline.</u> ☼ <u>Silicone intubation of the lacrimal drainage system:</u> the tube remains in situ for 6-12 months. ☼ <u>Dacryo-Cysto-Rhinostomy (DCR).</u> ✎ <u>Treatment of Complications</u> (conjunctivitis).	✎ <u>Treatment of Cause:</u> ☼ <u>NLD obstruction:</u> e.g. relieve congestion, remove of a nasal polyp, etc. ☼ <u>Infections:</u> anti-microbials ✎ <u>Treatment of Condition and lacrimal complications:</u> ☼ <u>Dacryo-Cysto-Rhinostomy (DCR):</u> ➤ Idea: Creation of opening between lacrimal sac and nasal mucosa of the middle meatus. ⇒ drainage of tears directly into the nose bypassing the NLD. ➤ Indications: Chronic dacryocystitis – Mucocele of the lacrimal sac –Lacrimal fistula (with fistulectomy). ➤ Contraindications: Bad sac e.g. in extensive adhesions – Swollen sac e.g. in TB and tumors –Bad nasal mucosa e.g. in atrophic rhinitis – Hypopyon CU. ☼ <u>Dacryo-cystectomy:</u> if DCR is contraindicated ✎ <u>Treatment of ocular complications:</u> e.g. conjunctivitis	✎ <u>Treatment of Cause:</u> ☼ <u>NLD obstruction:</u> e.g. relieve congestion, remove of a nasal polyp, etc. ☼ <u>Infections:</u> anti-microbials ✎ <u>Treatment of Condition:</u> ☼ <u>Hot fomentation</u> to decrease the pain. ☼ <u>Lotions:</u> to clean the pus. ☼ <u>Incision and drainage:</u> if an abscess forms. ✎ <u>Treatment of lacrimal complications:</u> ☼ <u>Dacryo-Cysto-Rhinostomy (DCR):</u> ☼ <u>Dacryo-cystectomy:</u> ✎ <u>Treatment of orbital complications:</u> e.g. orbital cellulitis and CST.

Conjunctiva

Conjunctivitis "general rules"

Definition: ✎ Inflammation of the conjunctiva.

Etiology (Classification):

⊙ Infective:

- ✎ Acute e.g. Viral, Muco-purulent, Purulent and Membranous conjunctivitis.
- ✎ Chronic e.g. Catarrhal, Angular, Granular and Inclusion conjunctivitis.

⊙ Non- infective:

- ✎ Acute e.g. Mechanical, Photophthalmia and Allergic conjunctivitis.
- ✎ Chronic e.g. Catarrhal, Phlyctenular, Spring catarrh and Giant papillary conjunctivitis.

Clinical picture (Pathological manifestations): see later:

Complications:

- ⊙ Corneal Complications: in most cases.
- ⊙ Corneal, Conjunctival, Lid and Lacrimal Complications: in cases with fibrosis e.g. Membranous and Granular conjunctivitis

Treatment:

- ⊙ Prophylaxis: by using protective measures against possible risk factors
- ⊙ Treatment of the Cause: e.g.
 - ✎ Infective conjunctivitis: by Anti-Microbials.
 - ✎ Allergic (non-Infective) conjunctivitis: by Anti-allergic medications.
- ⊙ Treatment of the Condition: by Frequent washing with Sterile water or Boric acid lotion 4%: to remove discharge.
- ⊙ Treatment of the Complications: ✎ CU: by Atropine, dark glasses & hot fomentation

Pathological manifestations:

📖 General manifestations: occur in most types of conjunctivitis

- ⊙ Hyperemia: injection of the posterior conjunctival vessels .
- ⊙ Chemosis: edema of the sub-epithelial CT (may be associated with lid edema).
- ⊙ Discharge: may be serous "watery", mucoid, mucopurulent or purulent depending on the etiology and severity of the inflammation.
- ⊙ Irritation: discomfort, itching, burning and foreign body sensation.

📖 Specific manifestations: occur in most types of conjunctivitis

⊙ Follicles:

- ✎ Definition: focal collections of lymphocytes in the substantia propria.
- ✎ Shape and Sites: gelatinous elevations most prominent in the inferior fornix.

✎ Causes: ✎ ✎

- Viral infections e.g. Adenovirus ,Herpes and M. contagiosum.
- Chlamydial infections e.g. Trachoma and adult inclusion conjunctivitis.
- Drug Allergy e.g. Atropine and glaucoma medications. ➤ Folliculosis.

⊙ Papillae:

- ✎ Definition: epithelial proliferation with a vascular core.
- ✎ Shape and Sites: multiple red bumps each with a central core of vessels usually seen at superior tarsal conjunctiva.

✎ Causes: ✎

- Bacterial infections e.g. Purulent conjunctivitis.
- Chlamydial infections e.g. Trachoma. ➤ Allergic cases e.g. Spring catarrh.

⊙ Giant papillae:

- ✎ Definition: giant papillae formed from union of multiple small papillae after rupture of fibrous septa attaching them to the underlying tarsus.

- ✎ Causes: ✎ ➤ CL conjunctivitis (giant papillae conjunctivitis).

⊙ Membranes:

- ✎ Color and Structure: dirty grayish formed of fibrin, inflammatory cells, necrotic cells and serous fluid coming from exudation of damaged vessels.

- ✎ On Removal ⇨ raw bleeding surface ⇨ adhesions and fibrosis

✎ Causes: ✎

- Diphtheric "membranous" conjunctivitis.
- Alkali or Acid burns "chemical trauma". ➤ Severe viral infections.

⊙ Pseudo-Membranes:

- ✎ Structure: formed by condensation of discharge and exudates.

- ✎ On Removal ⇨ intact epithelial surface "easily removed".

Conjunctival injection vs Ciliary injection

	Conjunctival injection	Ciliary injection
Origin	Posterior conjunctival vessels	Anterior ciliary vessels
Site	More marked at the fornix	More marked at the limbus
Appearance	Superficial and seen clearly	Deep and blurred.
Color	Bright red	Pink
Movement	Moves freely with conjunctiva	Can't move with conjunctiva
Differential test	Constricted by adrenaline	Not constricted by adrenaline
Causes	✎ Conjunctivitis	✎ Keratitis. ✎ Irido-cyclitis.

	Viral	Muco-purulent	Purulent (adult type)	Membranous (Diphtheric)
Definition	Acute infective inflammation of the conjunctiva caused by			
Etiology	- Adenovirus: spreads by droplet route causing epidemic kerato-conjunctivitis and may be accompanied by sore throat. - Herpes simplex, Enterovirus, Newcastle disease viruses, Influenza, Measles and Mumps.	- Haemophilus Aegypticus (Koch-Week's bacillus): the most common causing epidemics in April, May, September, and October. - Staphylococci, Streptococci and Pneumococci.	- Gonococci: 60-80% of cases. - Staphylococci, Streptococci and mixed infections. - the organism is transmitted through: Flies, dust, contaminated towels or through the genitals.	Corynebacterium diphtheriae: (rare nowadays as children are now effectively immunized).
Clinical manifest.	General: as general rules but: - Hyperemia may be associated with subconjunctival hemorrhage in acute hemorrhagic conjunctivitis. - Discharge is usually watery but may be purulent or mucopurulent. Specific: as general rules but: - Enlarged tender pre-auricular LN. - Follicle formation: "describe" - Vesicle formation: on the eyelid in herpetic cases.	General: as general rules but: - Hyperemia may be associated with petechial hemorrhage in severe cases. - Discharge is Mucopurulent formed of a mixture of mucus, tears, leucocytes and serum ⇒ gluing of lashes especially in the morning on waking up. Specific: No specific manifestations	Incubation period: no clinical manifestations for few hours to 3 days Stage of infiltration: General: as general rules but: - Hyperemia is marked. - Discharge is watery or mucoid. Specific: - Enlarged tender Pre-auricular LN. Stage of infiltration: General: as general rules but: - Chemosis is decreased "lids are tense". - Discharge is profuse and purulent. Specific: - Papillae formation: at the palpebral conjunctiva (velvet-like).	Incubation period: no clinical manifestations for 12 hours to 3 days Stage of infiltration: General: as general rules but: - Chemosis is covered with yellowish exudation. - Discharge is scanty & mucopurulent Specific: - True membrane: "as general rules". Stage of infiltration: General: as general rules but: - Hyperemia is marked. - Discharge is purulent blood-stained with pieces of the sloughed membrane. Specific: - True membrane: "fate as general rule" Systemic manifestations: FAHM, Infection of throat and naso-pharynx.
Comp.	Disappearance: spontaneous in 7-14 days. Spread to the fellow eye: few days after the first eye Corneal Complications: - Punctate epithelial keratitis: especially in epidemic kerato-conjunctivitis.	Corneal Complications: - CU: usually superficial crescentic and marginal.	Disappearance: spontaneous within 2 weeks or with treatment Chronicity: with mild residual hyperemia and chemosis Corneal Complications: - CU: central, marginal or may form ring ulcers ⇒ irido-cyclitis and perforation. - Respiratory failure. - Toxic nephritis - Neurological manifestations e.g. Neuropathy, accommodation paralysis and paralytic squint.	Ocular: due to fibrosis - Corneal complications: xerosis, corneal ulcers and vascularization. - Conjunctival complications: xerosis, symblepharon and pseudo-ptyerygium. - Lid complications: trichiasis, entropion, and symblepharon. - Lacrimal complications: xerosis (due to fibrosis of ducts & accessory gland). Systemic: due to diphtheric exotoxin - Toxic myocarditis and Heart failure.

Treatment	- Prophylaxis: by: - Use of separate towels. - Protecting the fellow eye. - Changing and boiling bed sheets. - Treatment of the Cause: Anti-viral - Example: topical Acyclovir (Zovirax) eye ointment. - Treatment of the Condition and Complications: <i>as general rules.</i>	- Prophylaxis: by: - Use of separate towels. - Protecting the fellow eye. - Combating flies and good hygiene. - Treatment of the Cause: Anti-biotics - Examples: Gentamycin, Tobramycin, Terramycin, Sulfacetamide 10%, Chloramphenicol and Quinolones - Route and dose: - Local drops: frequently at day. - Local ointment: at night because it has long acting, prevents gluing of lashes and allows exit of discharge - Systemic: in severe cases. - Treatment of the Condition and Complications: <i>as general rules but:</i> - Don't use bandage as it accumulates the discharge and allows more multiplication of the organism.	- Prophylaxis: by: - Use of separate towels. - Protecting the fellow eye. - Changing and boiling bed sheets. - Combating flies and good hygiene. - Healthy sex habits and Careful disinfection of infected fingers. - Treatment of the Cause: Anti-biotics - Examples: Gentamycin, Tobramycin, Terramycin, Sulfacetamide 10%, Chloramphenicol and Quinolones - Route and dose: - Local drops: frequently at day. - Local ointment: at night? ☺ - Systemic: single dose of Ceftriaxone is used for gonococcal cases. - Treatment of the Condition and Complications: <i>as general rules but:</i> - Don't use bandage and hot foment.	- Prophylaxis: by: - Immunization against diphtheria. - Isolation of the patients and notification of the health authorities. - Treatment of the Cause: Anti-biotics and Anti-Diphtheric serum - Anti-biotics route and dose: - Local ointment and eye drops (10000 u/ml). - Systemic IM injections - Anti-Diphtheric serum route and dose: - Local eye drops - Systemic: (40,000-60,000 U) can be repeated every 12 hours to neutralize the circulating toxin - Treatment of the Condition and Complications: <i>as general rules plus:</i> - Complete bed rest.
	Ophthalmia neonatorum (Infant type of purulent conjunctivitis)			
	Chronic Catarrhal Conjunctivitis			
	Definition: Any form of conjunctivitis occurring in the first month of life. ⚠ N.B. Any discharge of a newborn infant is suspicious since tears aren't secreted at this date. Etiology: - Bacterial: Gonococci, Staphylococci, Streptococci and Pneumococci. - Viral: Herpes simplex and Adenoviruses. Chlamydia. - The organism is transmitted through: ocular contact with contaminated maternal passages or towels. Clinical picture (stages): the same as adult type. Investigations: - Conjunctival smear: may show bacteria or inclusion bodies in chlamydia and HSV. - Immuno-fluorescent tests: for chlamydia. Treatment: Prophylaxis: - Before labor: treatment of the mother (especially herpetic cervicitis). - Just after labor: washing of the body of the baby from above downward. - After labor for 1 week: penicillin or broad spectrum antibiotic eye drops. - Treatment of the Cause, Condition and Complications: the same as adult type.			

Trachoma (Granular Kerato-Conjunctivitis)	
Definition: ✎ Combined inflammation of the conjunctiva and the cornea caused by Chlamydia trachomatis. ☠ N.B. Trachoma is the greatest single cause of preventable blindness in the world and It's endemic in Egypt affecting more than 80% of the population.	
Etiology: ◎ Predisposing factors: low socioeconomic status, childhood and endemic areas. ◎ Causative organisms: Chlamydia trachomatis which has the following characters: ☼ Large-sized obligate intracellular organism. ☼ Has a cell wall and Contains both DNA and RNA. ☼ Produces intracellular basophilic inclusion bodies in epithelial cells. ☼ Susceptible to Tetracycline, Erythromycin and Sulphonamides. ☼ No solid immunity so recurrences are common. ◎ Route of infection: through conjunctival discharge carried by fingers, towels and flies.	✎ Trachomatous pannus formation: ➤ Description: blood vessels running between the follicles (due to sub-epithelial cellular "lymphoid" infiltration and superficial vascularization). ➤ Course: ☼ Progressive pannus: Infiltration precedes vascularization which is parallel and directed vertically downward. ☼ Regressive pannus: Infiltration regresses and Vessels narrow. ☼ Healed pannus. ➤ Fate: ☼ Complete resolution: with no opacification if the BM isn't destroyed. ☼ Pannus siccus: with opacification if the BM is destroyed. ✎ Superficial keratitis: Numerous epithelial erosions involving the upper part of the cornea ⇒ positive staining with fluorescein ✎ Corneal ulcers: which may be: ➤ Typical trachomatous ulcer: ☼ Description: superficial, linear, horizontal at the lower edge of the pannus. ☼ Course: chronic, slowly spreading. ☼ Fate: healing with facet formation. ➤ Atypical trachomatous ulcers: ☼ Description: Marginal or central unrelated to the pannus. ☼ Cause: trachoma complications e.g. trichiasis.
Clinical picture: 📖 Conjunctival manifestations: "mainly in the upper palpebral conjunctiva" ◎ General manifestations: as general rules but mild and little. ◎ Specific manifestations: (Simplified MacCallan's Classification) ✎ T₁: Immature follicles formation: grayish small pin point not raised above the surface and non-expressible (due to sub-epithelial cellular infiltration around invading organism). ✎ T_{2-a}: Mature follicles formation: pinkish large raised above the surface & expressible. ✎ T_{2-b}: Papillae formation: pinkish soft and vascular. ✎ T₃: Healing by fibrosis (cicatrization): in the form of ➤ Patches or lines e.g. Arlt's line "dense white line at the sulcus sub-tarsalis" ➤ Post trachomatous degenerations (PTDs) between adjacent papillae. ➤ Post trachomatous concretions (PTCs) white calcified degenerations. ✎ T₄: Healed trachoma: end point with no follicles, papillae or inclusion bodies in conjunctival scraping.	✎ Xerosis: caused by: ➤ Atrophy of goblet cells. ➤ Fibrosis of the lacrimal ducts ⇒ obstruction. ➤ Fibrosis of lacrimal glands. ✎ Keratectasia: bulging of a thin weak corneal scar "cicatrix". ☠ N.B. Sure diagnostic clinical signs are Expressible follicles, Arlt's line, Post trachomatous degenerations (PTDs) and Pannus with Herbert's pits.
Complications: the same as ocular complications of membranous conjunctivitis plus: ➤ Epiphora may occur: due to chronic canaliculitis and chronic dacryocystitis.	
Investigations: ✎ ✎ Conjunctival scrapings stained with Giemsa stain: Intracytoplasmic basophilic inclusion bodies. ✎ Immunologic tests. .	
Treatment: ◎ Prophylaxis: ✎ ✎ Use of separate towels by patients. ✎ Combating flies and good hygiene. ✎ Careful disinfection of infected fingers. ◎ Treatment of the Cause: ABs (local drops and ointment for 6-12 weeks and Systemic) ✎ Examples: Sulfonamide, Tetracycline, Erythromycin and Quinolones. ◎ Treatment of the Condition: ✎ ✎ ✎ Expression of follicles. ✎ Scraping of papillae. ✎ Picking of PTDs. ◎ Treatment of the Complications: the same as general rules.	

Inclusion (swimming pool) Kerato-Conjunctivitis		Etiology:	
Definition: 🔪 Combined inflammation of the conjunctiva and the cornea caused by Chlamydia trachomatis type D.		🔪 <u>Predisposing factors:</u> as general rules of blepharitis.	
Etiology:		🔪 <u>Causative organisms:</u> Morax Axenfield diplo-bacilli (which secrete proteolytic enzymes ⇒ maceration of the epithelium).	
☉ <u>Causative organisms:</u> Chlamydia trachomatis type D.		💀 N.B. inflammation usually starts at lateral angles due to relative tear deficiency ⇒ absence of lysosomes which can inhibit the harmful proteolytic enzymes.	
☉ <u>Route of infection:</u> from the genitals by fingers or through water of swimming pools.			
Clinical picture:		Clinical picture:	
📖 Conjunctival manifestations: "mainly in the lower palpebral conjunctiva"		📖 Conjunctival manifestations:	
☉ <u>General manifestations:</u> as general rules but mild and little.		☉ <u>General manifestations:</u> as general rules of conjunctivitis but:	
☉ <u>Specific manifestations:</u> Follicles formation which Heal within 3-12 months.		🔪 <u>Discharge</u> is little white foamy. ☉ <u>Specific manifestations:</u> No specific manifestations.	
📖 Corneal manifestations:		📖 Eye-lid manifestations: especially at angles	
☉ <u>General:</u> ocular pain, photophobia, blepharo-spasm, lacrimation and ↓ed vision.		☉ <u>Margin:</u> red fissured and edematous. ☉ <u>Skin:</u> macerated and excoriated.	
☉ <u>Specific:</u> Superficial punctate keratitis.			
📖 Genital manifestations: ☉ Urethral or vaginal discharge.		Complications: 🔪 <u>Corneal Complications:</u> CU: Marginal and Hypopyon ulcers.	
Treatment: .		Treatment: ☉ <u>Prophylaxis:</u> as general rules of blepharitis	
☉ <u>Treatment of the Cause:</u> the same as Trachoma – the only line of treatment.		☉ <u>Treatment of the Cause:</u> Anti-biotics	
		🔪 <u>Examples - Route:</u> Tetracycline or Erythromycin – local (ointment) or systemic (oral)	
		☉ <u>Treatment of the Condition:</u> as general rules of conjunctivitis plus	
		🔪 <u>Vasoconstrictor eye drops:</u> e.g. Zinc sulphate aiming to Neutralize the action of the proteolytic enzyme. ☉ <u>Treatment of the Complications:</u> as general rules	
	Phlyctenular Kerato-Conjunctivitis		Vernal Kerato-Conjunctivitis (Spring catarrh)
Definition	🔪 Combined inflammation of the conjunctiva and the cornea caused by		
Etiology	🔪 <u>Hypersensitivity to an endogenous antigen:</u> e.g.		🔪 <u>Hypersensitivity to airborne (exogenous) allergens.</u>
	➤ Tuberculous proteins.	➤ Intestinal parasites.	💀 N.B. Spring catarrh has seasonal variation (in spring and summer) affecting mainly children and young adults (5-25 y) and more common in patients with asthma, hay fever, atopy and may be associated also with keratoconus.
	➤ Septic focus as tonsillitis.		
Clinical picture	📖 Conjunctival manifestations:		
	☉ <u>General manifestations:</u> as general rules but:		
	🔪 <u>Discharge</u> is watery or mucoid.		
	☉ <u>Specific manifestations:</u> phlycten 👉👈		
	🔪 <u>Color:</u> Grayish or yellowish.		
	🔪 <u>Shape:</u> rounded raised nodule.		🔪 <u>Size:</u> 1-3 mm in size.
	🔪 <u>Site:</u> common at the limbus and bulbar conjunctiva.		
	🔪 <u>Structure:</u> lymphocytic aggregation covered with intact epithelium, which ulcerates later with secondary infection.		
	🔪 <u>Surrounded by:</u> small area of congestion.		
	📖 Conjunctival manifestations:		
	☉ <u>General manifestations:</u> as general rules but:		
	🔪 <u>Discharge</u> is scanty whitish ropy mucoid 🔪 <u>Itching:</u> is severe and characteristic		
	☉ <u>Specific manifestations:</u> 👉 1. Palpebral Type: Papillae formation 👉👈		
	🔪 <u>Color:</u> bluish-white or red.		
	🔪 <u>Shape:</u> flat-topped giving a cobblestone appearance.		🔪 <u>Size:</u> large.
	🔪 <u>Site:</u> affecting mainly the upper tarsus (absent from the fornix)..		
	🔪 <u>Structure:</u> central core of connective tissue rich in eosinophils covered by thick epithelium with tiny twigs of blood vessels in the center and edges.		
	🔪 <u>On exposure (lid eversion):</u> sticky milky white discharge rich in eosinophils.		

Clinical picture	📖 Corneal manifestations: ⦿ General: pain, photophobia, blepharo-spasm, lacrimation and ↓ed vision. ⦿ Specific: 🔪 Corneal Phlycten formation: superficial or deep to BM. 🔪 Phlyctenular pannus formation: thin affecting any part of the limbus: ➤ Description: blood vessels running straight to BM. (due to cellular "lymphoid" infiltration and vascularization with rounded edge). ➤ Associated with: eczema of the lids and face, fissures at the outer canthus. 🔪 Corneal ulcers: which may be: ➤ Limbal ulcer: single or multiple which may fuse to form a ring ulcer. ➤ Fascicular ulcer: 🦋 ☁ Description: superficial creeping in a serpiginous manner to the center. ☁ Supplied by: leash of blood vessels. ☁ On healing: its track leaves an opacity maximum where it stops..	2. Bulbar type: gelatinous masses (⚠ N.B. more severe) 🔪 Site: starts at the upper limbus, then later all round. 🔪 Structure: connective tissue core with hyaline degeneration covered by hypertrophied (thick) epithelium. 🔪 May be associated with: white spot concretions of eosinophils and necrotic epithelium (Tranta spots). 3. Mixed type. 📖 Corneal manifestations: ⦿ General: pain, photophobia, blepharo-spasm, lacrimation and ↓ed vision. ⦿ Specific: 🔪 360-degree corneal pannus. 🔪 Fine punctate epithelial keratitis: 🔪 Corneal ulcers (rare).	
	Comp.	🔪 Corneal complications: (= corneal manifestations). 🔪 Conjunctival complications: 2^{ry} infection ⇒ mucopurulent conjunctivitis.	🔪 Corneal complications: (= corneal manifestations).
	Treatment	⦿ Prophylaxis and Treatment of the cause: if possible ⦿ Treatment of the condition: as general rules plus 🔪 Topical steroids. ⦿ Treatment of the Complications: 🔪 In cases with corneal affection: atropine and dark glasses. 🔪 In cases with Fascicular ulcer: cautery with carbolic acid and actual cautery for blood vessels at the limbus. 🔪 In cases with mucopurulent conjunctivitis: lotions and local antibiotics.	⦿ Prophylaxis: by Avoiding exposure to allergens if known. ⦿ Treatment of the Condition: as general rules plus 🔪 Mast cell stabilizers: ➤ Examples: Disodium chromoglycate and Lodoxamide. ➤ Mechanism: prevents mast cell degranulation ⇒ prevents histamine release 🔪 Vasoconstrictor eye drops: e.g. Zinc sulphate. 🔪 Anti-histaminic drugs: local eye drops and systemic. 🔪 Topical steroids: in severe non-responsive cases. 🔪 Beta irradiation: in more severe resistant cases. ⦿ Treatment of the Complications: as general rules (Atropine and Dark glasses) but 🔪 Cold compressors is more preferred than hot fomentations.
		Giant papillary conjunctivitis	
Definition and Etiology: 🔪 chronic non-infective conjunctivitis caused by: ⦿ Irritation of the conjunctiva: due to: ➤ Contact lens: "soft >hard & extended-wear > daily-wear". ➤ Ocular prosthesis. ➤ Exposed corneal sutures.		⦿ Specific manifestations: Giant papillae formation 🔪 Shape: flat-topped and covered with mucous. 🔪 Size: large. 🔪 Site: affecting mainly the superior tarsal conjunctiva.	
Clinical picture:		Treatment: ⦿ Treatment of the cause: 🔪 Discontinuation of use of the offending CL ⇒ symptoms resolve but the papillae will persist for several months. ⦿ Treatment of the condition: as spring catarrh	

	Eyelid		
	Squamous blepharitis	Ulcerative blepharitis	Parasitic blepharitis
Definition	✎ Chronic inflammation of the lid margin caused by ⚠ N.B. it's one of the most common external eye disorders in clinical practice.		
Etiology	📖 Predisposing factors: different irritants which cause hyperemia of the lid margin ✎ <u>Exogenous</u> : dust, wind, heat and smoke. ✎ <u>Endogenous</u> : ➤ <u>Local eye strain</u> e.g. Errors of refraction, Muscular imbalance, Over-work of fine nature, Over-work in poor illumination, and Loss of sleep. ➤ <u>Systemic factors</u> e.g. Allergic reactivity including the lid margin., Metabolic disturbances e.g. excess carbohydrate in diet, Dyspepsia, Hepatic insufficiency and DM. ➤ <u>Seborrhea, dandruff of the scalp and rosacea.</u>		
	📖 Causative organisms (Exciting Factors): ✎ Coryne-bacterium acne. ⚠ N.B. Zeis glands secrete excessive amount of neutral lipids ⇒ splitting by Coryne- bacterium acne into irritating free fatty acids ⇒ scales between lashes.	📖 Causative organisms (Exciting Factors): ✎ Staphylococcus aureus.	📖 Causative organisms (Exciting Factors): ✎ Phthirus pubis (Pubic lice).
Symptoms	✎ Itching and Burning sensation. ✎ Photophobia and Lacrimation.		
Signs	⦿ <u>Scales</u> : ✎ <u>Site</u> : between lashes. ✎ <u>Size and Color</u> : small white dried scales. ✎ <u>On removal</u> : reveals a hyperemic lid margin without ulceration.	⦿ <u>Crusts</u> : ✎ <u>Site</u> : at the base of lashes gluing them together. ✎ <u>Size and Color</u> : larger yellow crusts. ✎ <u>On removal</u> : reveals minute ulcers of the lid margin which bleed easily.	⦿ <u>Nits</u> : ✎ <u>Site</u> : covering the lashes.
Comp. of ulcerative blepharitis	✎ <u>Tylosis</u> : Thickening and hypertrophy of the lid margin. ✎ <u>Trichiasis</u> : due to healing of the ulcers by fibrosis. ✎ <u>Chronic conjunctivitis</u> . ✎ <u>Punctate keratitis</u> : in the lower one third of the cornea. ✎ <u>Marginal corneal ulcer</u> . ✎ <u>Madarosis</u> : due to destruction of hair follicles ✎ <u>Epiphora</u> : due to destruction of the sharp posterior lid margin initiating the vicious circle of epiphora ⇒ eczema ⇒ ✎ <u>Ectropion</u> ⇒ epiphora.		
Treatment	⦿ <u>Prophylaxis</u> : by caring of the predisposing factors e.g. ✎ Attention to the general health. ✎ Change of unhygienic atmosphere. ✎ Correction of any refractive errors ✎ Avoidance of excess carbohydrates in diet. ✎ Treatment of seborrhea of scalp.		
	⦿ <u>Treatment of the Cause</u> : ✎ Antibiotic ointment (rubbed into the lid margin). ⦿ <u>Treatment of the Condition</u> : ✎ Removing scales by 3% sodium bicarbonate or diluted baby shampoo.	⦿ <u>Treatment of the Cause</u> : ✎ Antibiotic ointment (rubbed into the lid margin). ⦿ <u>Treatment of the Condition</u> : ✎ Removing crusts by 3% sodium bicarbonate or diluted baby shampoo. ✎ More lid hygiene by frequent massage to evacuate Meibomian secretions. ⦿ <u>Treatment of the Complications</u> : if present	⦿ <u>Treatment of the Cause</u> : ✎ Yellow oxide of mercury 1% ointment: (rubbed into the lid margin). ⦿ <u>Treatment of the Condition</u> : ✎ Removing nits by diluted Acetic acid 2%. ✎ Cutting the lashes may facilitate the treatment.
	⚠ N.B. Treatment should be prolonged as organisms are hidden in the hair follicles and Meibomian glands.		

	Chalazion	Stye (Hordeolum Externum)
Definition	✎ Chronic non-specific inflammatory granuloma of Meibomian glands.	✎ Acute suppurative inflammation of Zeis gland and the lash follicle forming small abscess.
Etiology (Types)	✎ Obstruction of Meibomian glands ducts of Unknown etiology ⇒ retaining of secretions which are irritant and excite a granulomatous reaction. ☠ N.B. ₁ Possible causes of obstruction may include dry secretions and epithelial desquamation and proliferation caused by vitamin A deficiency. ☠ N.B. ₂ The granuloma contains many giant cells.	📖 Predisposing factors: the same as blepharitis. 📖 Causative organisms (Exciting Factors): ✎ Staphylococcus aureus. ☠ N.B. Ulcerative blepharitis usually predispose to Stye.
Symptoms	✎ Swelling of the lid: of long duration. ✎ No pain: except if become infected.	✎ Swelling of the lid. ✎ Pain: severe and starts dull then becomes throbbing.
Signs	✎ Swelling of the lid. ➤ Felt under the skin. ➤ Not related to lashes. ➤ Single or multiple.	✎ Swelling of the lid. ➤ Close to the lid margin. ➤ Related to a lash. ➤ Points on the skin side.
Fates	☉ Spontaneous resolution: rare. ☉ Complications: ✎ If the chalazion is large: ptosis in upper lid and ectropion in lower lid. ✎ Projection to: skin, conjunctiva or lid margin (marginal chalazion in which the granulation tissue forms only in ducts ⇒ projection as a red nodule). ✎ Infection ⇒ Acute chalazion (Hordeolum intemum).	☉ Complications: ✎ Recurrence. ✎ Madarosis: if the lash follicle is destructed).
Treatment	☉ Treatment of the Cause: by Vitamin A. ☉ Treatment of the Condition: ✎ Small chalazion: local antibiotic and steroid preparation. ✎ Moderate or large chalazion: vertical incision and scraping through the conjunctival side. ✎ Multiple chalazion: combined excision of the tarsus and conjunctiva leaving the lower third of the tarsus (to avoid lid notching) then replacement by a mucous graft from the lip. ✎ Marginal chalazion: scraping from lid margin followed by diathermy. ✎ Recurrent chalazion of the same gland: excision biopsy to exclude malignancy ☉ Treatment of the Complications: if present	☉ Prophylaxis: the same as blepharitis (important to prevent the recurrence). ☉ Treatment of the Cause: ✎ Antibiotic ointment: local (eye drops and ointment) and systemic. ☉ Treatment of the Condition: ✎ Hot fomentations to relieve pain. ✎ Evacuation of pus when pointing occur by epilation of the related lash then horizontal incision to avoid gapping. ☉ Treatment of the Complications: e.g. Madarosis by artificial lashes.
Hordeolum internum		✎ Allergic(Angio-neurotic): due to local medication e.g. atropine or due to systemic vasomotor disturbances e.g. with menses.
Definition:	✎ Acute suppurative inflammation of the Meibomian gland.	☠ N.B. ₁ edema accumulates in lid owing to the looseness of its subcutaneous areolar tissue.
Etiology:	✎ Staphylococcus aureus (primary or secondary on top of a chalazion).	☠ N.B. ₂ DD of lid edema: emphysema, myxedema and dermatochalasis.
Eyelid edema		
Etiology:	✎ Traumatic: following direct traumas of surgeries.	Rubbing lashes
✎ Inflammatory (Active): due to inflammation of lid, conjunctiva, cornea, iris, etc.		Definition ✎ rubbing of 4 lashes or less against the cornea or conjunctiva ⇒ irritation
✎ Systemic (Passive): due to renal, cardiac diseases or thyrotoxicosis.		Treatment: ✎ epilation (temporary) or follicle destruction e.g. electrolysis (permanent)

	Lagophthalmos	Symblepharon	Trichiasis									
Definition	✂ Incomplete closure of the palpebral fissure when the lids are closed.	✂ Adhesions between the bulbar and the palpebral conjunctiva.	✂ Rubbing of more than 4 lashes against the cornea or the conjunctiva.									
Etiology	⦿ Local causes: ✂ Physiological: lack of orbicularis tone during sleep. ✂ Paralysis of OO e.g. facial palsy (bell's palsy). ✂ Paralytic ectropion. ✂ Proptosis. ✂ Scarring of the lid. ✂ Coloboma of the lid. ⦿ General causes: ✂ Severe illness and weakness with atony of muscles	✂ Post-operative e.g. pterygium operation. ✂ Post-inflammatory e.g. trachoma and diphtheria. ✂ Burns, caustics and ocular cicatricial pemphigoid. Types of symblepharon: ✂ Anterior: between lid margin and cornea. ✂ Posterior: at the fornix (e.g. in trachoma). ✂ Total: between whole lid and globe (e.g. in burns).	✂ Trachoma: commonest cause due to fibrosis distorting the hair follicles. ✂ Ulcerative blepharitis.									
Clinical picture and Comp.	⦿ Epiphora. ⦿ Corneal complications: ✂ Exposure keratitis (esp. during sleep in lower 1/3). 📌 N.B. The upper part of cornea is protected by the upper lid as the eyes roll up during sleep (Bell's phenomenon) ⦿ Conjunctival complications: ✂ Chronic conjunctivitis and Xerosis (dryness).	✂ Ankyloblepharon. ✂ Bad cosmetic appearance. ✂ Chronic conjunctivitis ✂ Diminution of vision in cases of corneal affection. ✂ Diplopia and Limitation of ocular motility. ✂ Exposure keratopathy.	⦿ Symptoms of corneal irritation: e.g. ✂ Foreign body sensation. ✂ Photophobia, Blepharo-spasm and Lacrimation. ⦿ Corneal complications: ⦿ Conjunctival: ✂ Recurrent CU ⇨ opacities. ✂ Conjunctival ulcer. ✂ Epithelial plaque. ✂ Epithelial plaque. ✂ Superficial vascularization. ✂ Chronic conjunctivitis									
Treatment	⦿ Treatment of the Cause: e.g. ✂ Bell's palsy: by steroids and facial massage: to help nerve regeneration ⦿ Treatment of the Condition: e.g. ✂ Narrowing the palpebral fissure: by ➤ Tarsorrhaphy (Lateral, Para-median or Median) – (Temporary or Permanent). ➤ Fascia-lata sling and silicone sling operation (in severe recurrent cases). ✂ Corneal protection: by: ➤ Glasses, contact lenses or eye drops during day. ➤ Ointment at night (before sleep). ⦿ Treatment of the Complications: e.g. CU and Conjunctivitis.	⦿ Treatment of the Cause: e.g. Trachoma. ⦿ Treatment of the Condition: ✂ Cut the adhesions. ✂ Mucous membrane graft: to cover the two opposite surfaces. ✂ Contact shell: until healing occurs. ⦿ Treatment of the Complications: e.g. CU and Conjunctivitis. ✂ Keratoplasty: usually needed if the cornea becomes opaque.	⦿ Treatment of the Cause: e.g. Trachoma. ⦿ Treatment of the Condition: surgical correction <table><tr><td></td><td>Trichiasis alone</td><td>with entropion</td></tr><tr><td>Upper / Superior lid</td><td>Van Millingen's operation</td><td>Snellen's operation</td></tr><tr><td>Lower lid</td><td colspan="2">Webster's operation.</td></tr></table> ✂ Principle of Van Millingen's operation: displacing the lashes away from the cornea by placing a buccal mucous membrane graft in the grey line. ✂ Principle of Webster's operation: straightening the tarsus and lengthen the palpebral conjunctiva by placing a buccal mucous membrane graft in an incision in the sulcus sub-tarsalis. ✂ Principle of Snellen's operation: ☼ Straightening of the deformed tarsus by wedge resection of tarsus. ☼ Eversion of the lid margin. ☼ Displacing the lashes away from the cornea ⦿ Treatment of the Complications: e.g. CU and Conjunctivitis.		Trichiasis alone	with entropion	Upper / Superior lid	Van Millingen's operation	Snellen's operation	Lower lid	Webster's operation.	
	Trichiasis alone	with entropion										
Upper / Superior lid	Van Millingen's operation	Snellen's operation										
Lower lid	Webster's operation.											

Ptosis

Definition: ✂ Dropping of the upper eyelid below its normal position.

✂ N.B. Normally: the upper eyelid covers the upper 1/6 of the cornea (2mm).

Etiology (Types):

📖 Congenital: **dates since birth and usually hereditary and bilateral**

⦿ Causes:

✂ Myogenic: due to poor development or absence of levator muscle.

✂ Neurogenic "Marcus-Gunn Jaw winking ptosis": due to rare abnormal association between the 3rd and 5th cranial nerves ⇒ upper lid elevation from the dropped position only on movement of the jaw (associative movement).

⦿ Types:

✂ Simple.

✂ Associated with other anomalies (lid deformities or congenital ophthalmoplegia).

✂ N.B. **Blepharo-phimosis** ⚡: congenital ptosis, epicanthus, telecanthus and blepharo-phimosis.

📖 Acquired: **usually unilateral and maybe**

⦿ Myogenic: due to:

✂ Systemic disease: e.g. myasthenia gravis and myotonic dystrophy.

✂ Local disease: e.g. chronic progressive external ophthalmoplegia.

⦿ Neurogenic: due to:

✂ 3rd cranial nerve lesion ⇒ Levator muscle paralysis.

✂ Sympathetic nerve lesion: ⇒ Muller's muscle paralysis.

✂ N.B. Horner's syndrome: occurs due to sympathetic nerve lesion and consists of ptosis, miosis, anhydrosis, enophthalmos, flushing of face and heterochromia iridis.

⦿ Pseudo-ptosis: due to loss of eyelid support as in phthisis bulbi, enophthalmos and empty socket.

⦿ Mechanical: due to increased weight of the eyelid as in multiple chalazia, tumors, syphilitic tarsitis, trachoma, severe palpebral spring catarrh, amyloid and hyaline degeneration of the tarsus and conjunctiva.

⦿ Senile (involutional): due to:

✂ Primary weakness of levator muscle:

✂ Aponeurotic defects (attenuation, dehiscence and disinsertion from the tarsus).

⦿ Hysterical: due to:

✂ Incidence: unilateral or bilateral in young females with emotional trouble.

✂ Characters:

☼ Trembling of the ptotic lid. ☼ No wrinkling of the forehead or eyebrow elevation.

☼ Emotional behavior of the patient.

⦿ Traumatic: due to:

✂ Injury of the levator muscle or its nerve supply (Myogenic or Neurogenic).

✂ Emphysema, hemorrhage or edema of eyelid (Mechanical effect).

✂ Traumatic scarring of skin or conjunctiva.

⦿ Aponeurotic: due to:

✂ Old age (senility). ☼ Blepharo-chalasis. ☼ Cataract and ocular surgeries.

✂ Direct local blunt trauma. ☼ Chronic lid edema.

Clinical picture and Complications:

✂ Bad cosmetic appearance of the dropping UL.

✂ Wrinkling of the skin of the forehead (as an attempt to overcome partial ptosis by contraction of the occipito-frontalis muscle)

✂ Elevation of eyebrows.

✂ Absence of superior palpebral furrow (in marked ptosis).

✂ Elevation of the chin "compensatory head tilt" (in pronounced bilateral ptosis)

⦿ Interference with vision (if the pupillary area is covered in severe ptosis).

⦿ Amblyopia ex anopsia (in congenital unilateral severe ptosis).

⦿ Anesthesia of upper corneal part (in congenital ptosis).

⦿ Partial or complete ophthalmoplegia (in paralytic ptosis)..

Evaluation: ✂ ✂ ✂ ✂ ✂

⦿ Visual acuity: amblyopia ex anopsia may be present.

⦿ Binocular vision.

⦿ Bell's phenomenon: i.e. upward rotation of the eye on lid closure.

⦿ Corneal sensations: anesthesia of upper corneal part may occur in congenital ptosis

⦿ Other associated congenital anomalies.

⦿ Degree of ptosis

✂ How to measure? in the 1^{ry} position, measure the distance from upper lid margin to:

☼ The lower limbus at 6 o'clock. ☼ The center of the pupil. ☼ The eyebrow.

✂ Degree:

☼ Mild: upper eyelid covers 3-4mm of the cornea (1-2mm ptosis).

☼ Moderate: upper eyelid covers 5mm of the cornea (3mm ptosis).

☼ Severe: upper eyelid covers 6mm of the cornea or more (4mm ptosis or more).

⦿ Degree of elevation of eyelid by the levator muscle: levator action may be:

☼ Excellent: greater than 10 mm. ☼ Good: 7-10 mm.

☼ Fair: 4-6 mm. ☼ Poor: 3 mm or less.

⦿ Degree of elevation of eyelid by the frontalis muscle.

⦿ Degree of elevation of eyeball (globe) by the superior rectus muscle.

⦿ Extra-ocular muscle state (ocular movements state).

⦿ Elevation of the chin (abnormal head tilt – torticollis). .

Treatment:  Treatment of the Cause: <i>in acquired ptosis e.g.</i> ◉ <u>Mechanical ptosis</u>: remove the heavy lesion as chalazia or tumors. ◉ <u>Myogenic ptosis</u>: prostigmine medication for Myasthenia gravis. ◉ <u>Neurogenic ptosis</u>: wait for 6 months to allow nerve regeneration then do surgical treatment of the condition. ◉ <u>Senile ptosis</u>: levator resection. ◉ <u>Aponeurotic ptosis</u>: levator aponeurosis repair  Treatment of the Condition: Surgical correction ◉ <u>Fasanella-Servat operation</u>:  Indication: mild ptosis with good levator function.  Principle: part of Muller's muscle is excised together with the upper border of tarsus through conjunctiva.		◉ <u>Levator resection operation</u>:  Indication: moderate ptosis with fair levator function.  Principle: resection and advancement of the levator muscle to strengthen it.  Variations: ♣ If trans-cutaneous ⇒ Everbuech's operation. ♣ If trans-conjunctiva ⇒ Blascovic's operation. ◉ <u>Frontalis (Brow) suspension operation</u>: e.g. Hess operation – better used in bilateral cases  Indication: severe ptosis with poor levator function.  Principle: The upper lid is suspended to the occipito-frontalis muscle using: ♣ Endogenous materials as fascia lata. ♣ Exogenous materials as prolene.  Disadvantage: corneal exposure.  Treatment of the Complications: e.g. ◉ <u>Amblyopia</u>: occlusion (patching) of the preferred eye.	
	Entropion		Ectropion
Definition	 Rolling inward of the lid margin and the tarsal plate towards the eyeball.		 Rolling outward of the lid margin and the tarsal plate away from the eyeball.
Etiology (Types)	◉ <u>Fibrotic (Cicatricial)</u>: due to fibrosis of the palpebral conjunctiva :  Post-inflammatory: e.g. Trachoma and Membranous conjunctivitis.  Post-traumatic: e.g. Direct traumas (mechanical injuries or chemical burns) and Surgery (operations of the lids). ◉ <u>Infantile</u>: in plump (fatty) infants due to increased subcutaneous fat in the cheeks and lids pushing the lower lid margin inwards ◉ <u>Senile (involutional)</u>: usually occurs in the lower lid and:  Caused by senility features which are: ♣ Loss of orbital fat. ♣ Loss of the subcutaneous elastic tissue of the lid. ♣ Loose redundant skin of the lid.  In presence of one of these factors: ♣ Slight increase in the tone of the orbicularis muscle ♣ Prolonged bandaging of the eyes. ◉ <u>Spastic</u>:  Caused by weak support of the lid e.g. enophthalmos (especially senile).  In presence of Blepharo-spasm e.g. prolonged bandaging of the eyes. ◉ <u>Mechanical</u>: usually occurs in the lower lid:  Caused by weak support of the lid e.g. enophthalmos, empty socket and shrunken globe.  In absence of Blepharo-spasm. ◉ <u>Congenital</u>: usually occurs in the whole lower lid.		◉ <u>Fibrotic (Cicatricial)</u>: due to fibrosis and contracture of the lower lid skin by:  Burns, trauma or tumor. ◉ <u>Paralytic</u>: due to Paralysis of the orbicularis oculi muscle by:  Lower motor neuron lesion of the facial nerve e.g. Bell's palsy.  Direct trauma to the muscle. ◉ <u>Senile (involutional)</u>: usually occurs in the lower lid and:  Caused by senility features which are: ♣ Loose redundant skin of the lid. ♣ Lax orbicularis muscle fibers and palpebral ligaments.  Aggravated by: ♣ Conjunctivitis. ♣ Epiphora. ◉ <u>Spastic</u>: occurs typically in children and young people  Caused by well-support of the lid with firm overlying skin e.g. proptosis and chronic conjunctivitis(thickening of the conjunctiva).  In presence of spasm of the orbicularis muscle. ◉ <u>Mechanical</u>: due to increased weight of lower lid by e.g. multiple chalazia. ◉ <u>Congenital</u>: rare.

Symptoms		✎ Constant Epiphora due to eversion of the lacrimal punctum ⇒ Eczematous changes in the skin ⇒ aggravation of the Ectropion ⇒ more Epiphora
Signs		✎ <u>Mild</u>: exposure of the lower punctum. ✎ <u>Moderate</u>: exposure of the tarsal conjunctiva. ✎ <u>Severe</u>: exposure of the lower fornix.
Comp.	✎ The same as trichiasis 😊 but more severe ☹ ⇒ permanent corneal opacities and visual deterioration.	⦿ <u>E vicious circle</u>: Epiphora ⇒ Eczema of the lid skin ⇒ more Ectropion. ⦿ <u>Lagophthalmos</u>: in severe cases ⦿ <u>Corneal complications</u>: ✎ Exposure keratitis. ✎ CU. ✎ Xerosis (dryness). ⦿ <u>Conjunctival complications</u>: ✎ Chronic exposure conjunctivitis ✎ Chronic conjunctival hyperemia. ✎ Hypertrophy and Xerosis (dryness).
Treatment	📖 Treatment of the Cause: e.g. ✎ Trachoma and membranous conjunctivitis. ✎ Eye bandages: by removing them 1st (i.e. before surgical correction). 📖 Treatment of the Condition: Surgical correction according to type ⦿ <u>Fibrotic (Cicatricial)</u>: <i>the same as mentioned in trichiasis</i> 😊 ✎ In the superior lid: Snellen's operation. ✎ In the lower lid: Webster's operation. ⦿ <u>Infantile</u>: disappears spontaneously after some time. ⦿ <u>Senile (involutional)</u>: ✎ <u>Permanent methods</u>: Wheeler's operation (orbicularis muscle overlap). ✎ <u>Temporary methods</u>: ➤ Painting the lid skin with collodion. ➤ Alcohol injection along the edge of the lid (1 cc of 70% alcohol S.C.). ➤ T-shaped adhesive plaster ➤ Lateral canthotomy (division of the lateral canthus with scissors). ⦿ <u>Spastic</u>: ✎ <u>Permanent methods</u>: ➤ Skin and muscle operation: removal of skin piece and spastic orbicularis muscle close to lid margin (Riolan). ➤ Lateral canthoplasty: division of the lateral canthus and covering the raw area with bulbar conjunctiva. ✎ <u>Temporary methods</u>: <i>the same as senile type</i> 📖 Treatment of the Complications: e.g. CU and Conjunctivitis.	📖 Treatment of the Cause: e.g. ✎ <u>Bell's palsy</u>: by steroids and facial massage: to help nerve regeneration. ✎ Conjunctivitis. ✎ Blepharo-spasm. ✎ <u>Mechanical lesions</u> e.g. <u>chalzia</u>: by excision.. 📖 Treatment of the Condition: Surgical correction according to type ⦿ <u>Fibrotic (Cicatricial)</u>: ✎ In small scars: V to Y plasty or Z plasty. ✎ In large scars: free skin graft. 💀 <u>N.B.</u> Free skin graft is taken from non-hairy area with color like that of the face e.g. other eyelid, behind the ear or inner side of the arm. ⦿ <u>Paralytic</u>: narrowing the palpebral fissure by: ➤ Lateral tarsorrhaphy (temporary or permanent). ➤ Fascia-lata sling and silicone sling operation: in severe recurrent cases. ⦿ <u>Senile (involutional)</u>: according to the degree of ectropion: ✎ <u>Mild degree</u>: ➤ Electrocautery punctures: to induce fibrosis at the site of electrocautery ⇒ pull the lid inwards. ➤ Snellen's sutures: to produce fibrotic bands long the tracks of the sutures ⇒ pull the lid inwards. ✎ <u>Advanced degree</u>: Dimmer's modification of Kuhnt Szymanowski's operation: to remove the redundant skin and do wedge resection of the tarsus.. ⦿ <u>Spastic</u>: manual correction of ectropion then support the eyelid by a well-fitting bandage as a temporary measure. (unless contraindicated) 📖 Treatment of the Complications: e.g. Lagophthalmos, CU and Conjunctivitis.

Ocular Trauma

Blunt trauma

Introduction:

- ✎ If a large object e.g. a football hits the eye, most of the impact is usually taken by the orbital margin.
- ✎ If a small object e.g. tennis ball hits the eye, the eye itself takes most of the impact.

Effects of blunt trauma on the orbit:

📖 Traumatic proptosis:

⦿ Mechanisms: 🖐️

1. Retro-bulbar hemorrhage:

- It occurs after direct trauma, orbital fractures or retro-bulbar injection

2. Surgical emphysema:

- Following trauma to the ethmoid sinuses ⇒ air passes into the orbit and subcutaneous tissues of the eyelids.
- It ↑ with blowing of the nose and gives the feeling of soft crepitations in the lid.

3. Arterio-Venous fistula (Carotid-Cavernous fistula):

- ✎ Following severe head trauma ⇒ the internal carotid artery ruptures where it passes in the cavernous sinus ⇒ severe rise of venous pressure

⦿ Clinical picture: 🖐️🖐️

1. Proptosis: pulsatile with audible bruit.
2. Redness and Chemosis.
3. Papilledema and Dilated retinal veins.
4. Ophthalmoplegia (paralysis of the EOMs).

📖 Traumatic enophthalmos (Blow-out fracture):

⦿ Mechanisms: 🖐️ it occurs after trauma which may increase the intra-orbital pressure

⇒ fracture of the thin orbital walls, especially the floor.

⦿ Clinical picture: 🖐️🖐️

1. Enophthalmos: due to herniation of fat into the maxillary sinus.
2. Peri-orbital emphysema (i.e. air in eyelids): due to communication between the orbit and the peri-orbital sinuses
3. Defective eye movement: usually elevation, due to entrapment of an extra-ocular muscle in the fractured site.
4. Diplopia.

Effects of blunt trauma on the Eyelids: 🖐️

1. Traumatic ptosis: due to:

- ✎ Injury of the levator muscle or its nerve supply (Myogenic or Neurogenic).
- ✎ Emphysema, hemorrhage or edema of eyelid (Mechanical effect).
- ✎ Traumatic scarring of skin or conjunctiva.

2. Ecchymosis of the eyelids (traumatic black eye): treated by:

- ✎ Cold compresses in the first 24 hours (to induce vasoconstriction ⇒ ↓ bleeding).
- ✎ Hot compresses after 24 hours (to help absorption).

3. Lid lacerations: may be:

- ✎ Horizontal wounds: don't gape and produce a small scar.
- ✎ Vertical wounds: gape and need suturing.

Effects of blunt trauma on the Conjunctiva: 🖐️

1. Chemosis: edema of the conjunctiva.

2. Conjunctival lacerations: sutured if large.

2. Sub-conjunctival hemorrhage (blood under the conjunctiva):

- ✎ Sub-conjunctival hemorrhage may result from ocular blunt trauma which lead to rupture of conjunctival vessels and may result from leaking of blood from a fracture of the base of the skull due to severe head injury:

	Local ocular trauma	Fracture base of skull
Site	Usually on the temporal side.	Usually in the fornices.
Shape	Triangular, base towards cornea	Triangular, apex towards cornea
Color	Ocular trauma.	Head trauma.
Cause	Bright red.	Dark red.
Consciousness	Normal	Lost.
Onset	Immediate	Delayed.
Posterior limit	Seen.	Not seen.

Effects of blunt trauma on the Sclera: Scleral rupture (Ruptured globe).

1. Site:

- ✎ Usually up and in (concentric with and about 3 mm behind the limbus) because the trauma usually comes from down and out (where the globe is least protected and the eyeball is pushed against the trochlea)

2. Clinical picture:

- ✎ Sudden diminution of vision with pain, watering and redness of the eye
- ✎ Chemosis (edema) of the conjunctiva and/or sub-conjunctival hemorrhage.
- ✎ Uveal prolapse (with or without vitreous loss).
- ✎ Hypotony (low ocular tension).
- ✎ Shallow AC (with or without Hyphema).
- ✎ Abnormal site, size, and shape of the pupil.
- ✎ Complicated by infections, sympathetic ophthalmitis, IOFB (as penetrating trauma).

3. Treatment: the same as penetrating trauma

Effects of Blunt trauma on the cornea:**1. Corneal rupture:**

✎ Incidence: less common than scleral rupture.

✎ Clinical picture and treatment: the same as scleral rupture.

2. Corneal edema: Due to trauma to the endothelium and Descemet's membrane.**3. Recurrent corneal erosions:**

✎ Caused by scratches with fingernails or paper ⇒ imperfect healing of the epithelial basement membrane ⇒ recurrent corneal erosions with any slight trauma such as opening the eyes in the morning.

4. Corneal abrasions:

✎ Clinical picture and Treatment: as any CU ☺ (i.e. Atropine + corneal protectors + ABs).

5. Corneal foreign bodies:

✎ Treatment: ✎ steps:

- Topical anesthesia. ➤ Removal using a foreign body spud.
- Topical cycloplegic, antibiotic drops and ointment with patching of the eye

6. Blood-staining of the cornea:

✎ Cause: traumatic Hyphema (blood in the AC due to ruptured vessels) with ↑ed IOP.

✎ Treatment:

- Treatment of the cause (i.e. Hyphema): ✎ ✎ ✎
 - ☼ Bed rest in a semi -sitting position.
 - ☼ No aspirin or non-steroidal anti-inflammatory drugs.
 - ☼ Oral anti-fibrinolytic e.g. Aminocaproic acid every 4 hours to avoid re-bleeding.
 - ☼ Daily measurement of the ocular tension.
 - ☼ Topical beta blockers to control IOP (Timolol or Betaxolol).
 - ☼ Topical steroids to control iritis.
- Treatment of the condition (i.e. Blood staining of the cornea):
 - ☼ Early stage (i.e. Hyphema with high IOP): immediate evacuation (paracentesis).
 - ☼ Late stage (i.e. blood-staining becomes permanent): keratoplasty.

Effects of blunt trauma on the anterior chamber: Traumatic Hyphema: as cornea ☺**Effects of blunt trauma on the uveal tract:**

📖 Effects on the iris: ✎ ✎

1. Iris sphincter tears: defects in the constrictor pupillae muscle at the pupillary border which appear clinically as small V -shaped tears at the pupillary border.

⚠ N.B. Mydriatics should be avoided since they enlarge the tears.

2. Irido-dialysis: separation of the root of the iris from the ciliary body which appear clinically as D-shaped pupil.

⚠ N.B. It's often associated with hyphema and patients complain from uniocular diplopia or glare

3. Traumatic aniridia: complete avulsion of the iris at its root.

4. Traumatic iritis inflammation of the iris.

📖 Effects on the pupil: ✎ ✎

1. Traumatic miosis: curs with milder trauma due to irido-cyclitis.

2. Traumatic mydriasis: occurs with more severe trauma due to paralysis of the third nerve fibers. It's associated with paralysis of accommodation ⇒ blurring of near vision.

3. Poor reaction to light: this indicates ed ICT in patients with associated head injury.

📖 Effects on the ciliary body: ✎ ✎ ✎

1. Traumatic cyclitis: inflammation of the ciliary body.

2. Cyclo-dialysis: separation of the ciliary body from the scleral spur ⇒ Hypotony.

3. Traumatic spasm of accommodation (cyclo-spasm): with temporary myopia.

4. Traumatic paralysis of accommodation.

5. Hypotony: due to suppression of the aqueous humor secretion.

6. Angle recession glaucoma: due to ciliary body injury near the angle.

📖 Effects on the choroid:

1. Spontaneous choroidal detachment: due to hypotony:

2. Traumatic choroiditis: inflammation of the choroid.

3. Choroidal effusion or hemorrhage.

4. Rupture of the choroid: linear rupture may occur concentric with the optic disc:

✎ Symptoms: asymptomatic except it's under the fovea ⇒ severe visual impairment.

✎ Signs: early, covered with hemorrhage – then later, white sclera is seen through it.

Effects of blunt trauma on the lens:

1. Traumatic cataract. 2. Traumatic lens displacement. discuss both form lens chapter ☺

Effects of blunt trauma on the Vitreous:

1. Vitreous hemorrhage. 2. Vitreous opacities or floaters. 3. Avulsion of its base ⇒ RD.

4. Vitreous prolapse through a ruptured globe with traction on the retina.

Effects of blunt trauma on the Retina:**1. Commotio retinae:**

✎ Cause: contre-coup injury to posterior pole ⇒ swelling of ganglion cells (i.e. edema).

✎ Symptoms: rapid drop in vision. ✎ Signs: Retinal opacification (usually grayish-white) with or without scattered retinal hemorrhages and cherry-red fovea.

✎ Fate: ➤ Spontaneous visual recovery within a month.
➤ Macular degeneration or macular holes with severe loss of vision.

2. Hemorrhages: Retinal (superficial or deep) or sub-hyaloid.

3. Retina tears and dialysis: lead to rhegmatogenous retinal detachment

4. Retinal detachment: rhegmatogenous (due to tears), exudative (due to hypotony) or tractional (due to vitreous prolapse and incarceration in a sclera wound)

Perforating (Penetrating) trauma	
Definition and Etiology: ✎ Trauma to the eye with a sharp object such as a nail, knife, needle, scissors, or a piece of glass ⇒ corneal or/and scleral lacerations. ✎ It may or may not be accompanied with an intraocular foreign body.	3. Treatment of sympathetic ophthalmitis: ✎ <u>Prevention:</u> very important ➤ If the traumatized eye is grossly damaged or has no hope for repair ⇒ enucleate it. ➤ If there is hope ⇒ excise prolapsed tissues, remove FBs and suture wounds. ➤ Bilateral post-operative regular examination to detect early signs of inflammation. ✎ <u>Treatment:</u> ➤ Corticosteroids and Atropine (i.e. the usual treatment of uveitis) for both eyes to ↓ the inflammation (prolonged for months for fear of recurrence) ➤ If there is no response in 2-3 weeks ⇒ enucleate the exciting eye to save the sympathizing eye. 3. Treatment of intra-ocular foreign bodies: Removal ✎ <u>Indication of removal:</u> all FBs (except inert FBs). ✎ <u>Methods of removal:</u> through pars-plana vitrectomy and removal with a FB forceps (except magnetic FBs which may be pulled out with a magnet).
Clinical effects and complications: 1. Mechanical effects: immediate effects – the same as ruptured globe plus: ✎ Wounds of the lids and conjunctiva. ✎ Traumatic cataract. ☠ N.B. The eye should be gently examined (i.e. direct pressure should be avoided for fear of rupturing a partial thickness wound). 2. Ocular infections: occur within 1-2 days (but milder fungal infections may be delayed). 3. Sympathetic ophthalmitis: after a long period (average 1-2 months up to several years). ✎ It refers to: bilateral specific diffuse inflammation of the entire uveal tract. ✎ Symptoms and signs: symptoms and signs of Irido-cyclitis (earlier in exciting eye). 4. Intra-ocular foreign bodies: with serious consequences depending on its type: ✎ <u>Siderosis bulbi:</u> with iron foreign bodies: ➤ Siderotic cataract: It is not a true cataract, but a rusty discoloration due to iron deposition in the sub-capsular epithelium. ➤ Heterochromia iridis: With the ipsilateral iris darker. ➤ Secondary open angle glaucoma: from scarring of the trabecular meshwork. ✎ <u>Chalcosis bulbi:</u> with copper foreign bodies: ➤ Sunflower cataract. ➤ Kayser-Fleisher ring: due to copper deposition in Descemet's membrane ⇒ a golden brown ring. ☠ N.B. Pure copper produces severe inflammation that simulates endophthalmitis while Copper in alloys binds specifically to collagen and basement membranes.	Sympathetic Ophthalmitis Definition and Etiology: ✎ Bilateral specific diffuse inflammation of the entire uveal tract, usually following perforating trauma or ruptured one eye (especially with retained intra-ocular FBs). ✎ Allergy to uveal pigment of the injured eye excites bilateral inflammation. Clinical picture: ✎ 1. History, symptoms and signs of ocular trauma (write in brief) 2. Latent period between the trauma and uveal inflammation: ➤ Average 1-2 months. ➤ may be short down to 10 days or long up to 30 years! 3. Symptoms and signs of uveal inflammation (earlier in exciting eye) (write in brief). Investigations and Treatment: the same as penetrating trauma. 😊
Investigations: ✎ Careful fundus examination, X-ray and CT scan to localize the foreign bodies Treatment: Treatment of the condition (i.e. clinical effects and complications) 1, 2. Treatment of mechanical effects and ocular infections: ✎ ✎ ✎ Immediate patching and prophylactic systemic antibiotics. ✎ Anti-tetanus toxoid may be given if the patient is not vaccinated. ✎ Anti-emetics may be given because vomiting is very dangerous with an open globe. ✎ Surgical repair of the wound with ➤ Reposition of the prolapsed uvea. ➤ Abcission (excision) of the prolapsed uvea (if the eye is badly damaged).	Intra-Ocular Foreign Bodies (IOFBs) Classification and Etiology: ☉ <u>Metallic foreign bodies:</u> most common – may be: ✎ Magnetic: e.g. iron. ✎ Non-magnetic: e.g. copper and lead. ☠ N.B. Metallic FBs enter the eyes of workers who operate high-speed grinders or those using a hammer and chisel without protection (Lead pellets are common from firearm injuries). ☉ <u>Non-Metallic foreign bodies:</u> e.g. glass. ☠ N.B. Glass foreign bodies usually result from car accidents or breakage of eye glasses. Clinical picture, Investigations and Treatment: the same as penetrating trauma. 😊

Chemical Injuries (Trauma – burns)

Definition and Etiology:

✎ Ocular injury with accidental or occupational exposure to chemicals which may be: ✎

1. Alkali burns:

✎ Examples: lime (CaO), KOH, NaOH, cement, plasters, aniline dyes and ammonia which are present in household detergents, fertilizers, and refrigerants.

✎ Characters: more severe than acid burns because of their rapid penetration through the cornea and anterior chamber often in less than one minute (as they combine with cell membrane lipids thereby resulting in disruption of the cells and necrosis of the tissues).

✎ N.B. Lime (CaO), when combined with water of tears and tissues it transforms to Ca(OH) resulting in severe heat, caustic effect penetrating deeply to the eye tissues.

2. Acid burns:

✎ Examples: battery fluid (sulfuric acid), laboratory glacial acetic acid and bleach.

✎ Characters: less progressive and less penetrating than alkalis producing their maximum damage within the first few minutes to hours (as they precipitate tissue proteins that rapidly set up barriers against deep penetration).

3. War gases:

✎ Examples: mustard gas.

✎ Characters: can cause severe keratitis and permanent corneal scar

Clinical picture (effects) and complications:

⊙ Symptoms: Pain, photopia, blepharo-spasm, lacrimation and ↓ed vision ☺

⊙ Signs: **according to the severity of exposure**

✎ In cases with mild to moderate exposure:

- Eyelid edema.
- Conjunctival edema (chemosis) and injection.
- Corneal abrasions.
- Anterior uveitis.

✎ In cases with severe exposure:

- eyelid symblepharon and lagophthalmos.
- Conjunctival and epi-scleral whitening (coagulative necrosis).
- Corneal edema and opacification with cornea-scleral melting.
- Severe uveitis.
- Secondary glaucoma.
- Posterior segment destruction.

Treatment:

📖 Treatment of the cause: **immediate emergency treatment** ✎

⊙ Captious irrigation with specific antidotes: 1st aid if the chemical nature is known e.g.

✎ Alkali burns: boric acid 4%, citric acid (lemon juice) or acetic acid (vinegar).

✎ Lime burns: Neutral ammonium tartarate 10%, Concentrated glucose 40% or saturated sugar solution.

✎ Acid burns: sodium bicarbonate 3%.

✎ Iodine burns: atropine, starch suspension or milk.

✎ Aniline (Hair) dye burns: tannic acid or glycerol or dilute alcohol.

✎ E.D.T.A 1% is a universal antidote.

⊙ Captious irrigation with water or saline: if the chemical nature is not known or the antidotes are not available.

✎ Amount and Duration: several liters should be used for at least 1 hour.

✎ Precautions: ✎

- In lime burn, picking of the lime particles must be done before irrigation to avoid excessive heat production.
- Irrigation should never be delayed for any reason.
- It's better to place an eye speculum and topical anesthesia before irrigation.
- The lower lid is pulled down and the upper lid is everted to irrigate the fornices.
- Conjunctival pH should be tested 10 minutes after cessation of the irrigation using litmus paper ⇒ irrigation should be continued until neutral pH is reached (7.4).

📖 Treatment of the condition: **according to the severity of exposure**

⊙ In cases with mild to moderate exposure:

✎ Analgesics: to relieve pain.

✎ Topical antibiotics, Cycloplegics: for corneal lesions.

✎ Topical steroids: to reduce the inflammation (i.e. uveitis) provided that corneal epithelium is intact, otherwise, it's contraindicated.

⊙ In cases with severe exposure:

✎ Debridement of necrotic tissue (i.e. cut the adhesions): for symblepharon.

✎ Tarsorrhaphy: for lagophthalmos.

✎ Limbal, conjunctival, and autograft transplants and even penetrating keratoplasty: for severe corneal damage (melting).

✎ Oral Acetazolamide (Diamox) or topical beta-blockers: for secondary glaucoma.

Physical Injuries

Definition, Etiology and Clinical effects:

✎ Ocular injury with exposure to physical irritants which include: ✎

1. Infra-red waves (longer wave-length): either:

- Occupational rays in glass blowers ⇒ cataract (prevented by protective goggles).
- Solar rays (especially with direct looking at solar eclipse): central retinal burn.

2. Ultra-violet rays (shorter wave-length) ⇒ Photophthalmia (snow blindness) ☺

3. Microwaves ⇒ cataract.

4. X-rays: therapeutic doses (i.e. large) ⇒ cataract (prevented by covering eyes).

5. Visual display units and television sets: ⇒ eye strain after several hours of exposure.

Final Revision Part (2)

Happy Ramadan with



Happy ophthalmology

Dr. AbdulRahman Al-Hosary

Errors of refraction p1

Neuro p18

Lens p4

Tumors p22

Glaucoma p9

Strabismus p23

Retina p14

Collections (wait soon)

Happy ophthalmology series:

📖 Happy Final revision (2 parts): ⇐ 😊

✂ Smart view of the whole curriculum in tables.

📖 Happy Exams with model answers:

✂ How to answer? ✂ Exams of previous years with answers (as whole exams and also arranged on chapters).

📖 Happy MCQs:

✂ Official MCQs arranged , organized and revised.
✂ Examples from exams of previous years with answers.

📖 Happy clinical and oral:

✂ Clinical tests. ✂ Clinical decision making.
✂ Oral notes.

"من سلك طريقا يلتمس فيه علما، سهل الله له طريقا إلى الجنة.. وإن الملائكة لتضع أجنحتها لطالب العلم رضا بما يصنع.. وإن العالم ليستغفر له من في السماوات ومن في الأرض حتى الحيتان في الماء.. وإن فضل العالم على العابد كفضل القمر ليلة البدر على سائر الكواكب.. وإن الأنبياء لم يورثوا دينارا ولا درهما وإنما ورثوا العلم.. فمن أخذه أخذ بحظ وافر"
صدق رسول الله - صلى الله عليه وسلم-

Errors of refraction																											
	Myopia		Hypermetropia		Astigmatism																						
Definition	Error of refraction in which: Parallel rays come to a focus in front of the retina.		Error of refraction in which: Parallel rays come to a focus behind the retina.		Error of refraction in which: Parallel rays doesn't come to a focus, instead, the eye produces image with multiple focal points or lines (i.e. the eye has different powers in different meridians).																						
	Provided that, accommodation is completely relaxed.																										
Etiology	Axial Myopia: due to increased axial length of the eye (long eye). Curvature Myopia: due to increased curvature of the cornea or the lens (e.g. in acute irido-cyclitis). Index Myopia: due to increased refractive index of the cornea or the lens (e.g. in nuclear cataract and hyperglycemia). Anterior lens dislocation.		Axial Hypermetropia: due to decreased axial length of the eye (short eye). Curvature Hypermetropia: due to decreased curvature of the cornea or the lens (e.g. cornea plana). Index Hypermetropia: due to decreased refractive index of the cornea or the lens (e.g. in immature cortical cataract and hypoglycemia). Posterior lens dislocation (Aphakia).		Corneal astigmatism: more common – may be due to: ➤ Surgical or traumatic scars. ➤ Ectatic diseases of the cornea as keratoconus. ➤ Corneal irregularity of unknown cause. Lenticular astigmatism: rare – may be due to: ➤ Lens subluxation. ➤ Tilted IOL.																						
	Blurring of far vision. Muscular asthenopia (e.g. burning sensation, headache, dry eye, lacrimation etc.). Screwing of eyelids to simulate a pinhole ⇒ increase the depth of focus.		Blurring of near vision. Accommodative asthenopia (e.g. burning sensation, headache, dry eye, lacrimation etc.).		Blurring of vision. Accommodative asthenopia (e.g. burning sensation, headache, dry eye, lacrimation etc.).																						
Signs	We can measure degree of refractive errors by objective or subjective means e.g. Manual retinoscopy: using a plain mirror and a source of light to produce a red reflex (after inducing cycloplegia by Cyclopentolate or Atropine). Automated refractometers: for rapidly determining objective refraction.																										
					Visual Acuity charts: patient reads some types on the charts and can't read other types on the same line Other special tools: e.g. placido disc, keratometer and corneal topography.																						
Types of Myopia and Astigmatism	<table><tr><td></td><td>Simple</td><td>High</td><td>Congenital</td></tr><tr><td>Onset</td><td>Around 14 years</td><td>Around 7 years</td><td>Since birth</td></tr><tr><td>Progress till</td><td>Less than 20 years</td><td>More than 20 years</td><td>Stationary</td></tr><tr><td>Degree</td><td>Less than -6 D</td><td>May reach -25 D</td><td>About -10 D</td></tr><tr><td>Complications</td><td>Absent</td><td>Present</td><td>Present but mild</td></tr></table>				Simple	High	Congenital	Onset	Around 14 years	Around 7 years	Since birth	Progress till	Less than 20 years	More than 20 years	Stationary	Degree	Less than -6 D	May reach -25 D	About -10 D	Complications	Absent	Present	Present but mild	Regular astigmatism: in which the strongest and the weakest meridians are perpendicular and meridians in between are regularly arranged – it may be : ☼ Simple: one meridian is emmetropic and the other is ametropic (i.e. simple myopic or simple hyper-metropic astigmatism). ☼ Compound: both meridians are ametropic but of the same type (i.e. compound myopic or compound hyper-metropic astigmatism). ☼ Mixed: one meridian is myopic and the other is hyper-metropic. Irregular astigmatism: in which the strongest and the weakest meridians aren't perpendicular and meridians in between aren't regularly arranged.			
		Simple	High	Congenital																							
Onset	Around 14 years	Around 7 years	Since birth																								
Progress till	Less than 20 years	More than 20 years	Stationary																								
Degree	Less than -6 D	May reach -25 D	About -10 D																								
Complications	Absent	Present	Present but mild																								
N.B. High myopia is hereditary, more common in females, and has a racial tendency.																											

Complications	Complications of Myopia: 1. Macular complications: e.g. ➤ Macular hole ➤ Macular hemorrhage. ➤ Fuch's spot. (black area at the macula ⇒ loss of central vision). 2. Consecutive optic atrophy. 3. Complicated cataract. 4. Temporal crescent. 5. Divergent squint. 6. Degeneration and Detachment (posterior) of the vitreous ⇒ floaters (Musca volitantes). 7. Degeneration (peripheral) and Detachment of the choroid and retina ⇒ night blindness. 8. Posterior staphyloma. 9. Primary open angle glaucoma (genetically linked). 10. Pigmentary glaucoma. 11. Lens subluxation and dislocation.		
	Complications of Hypermetropia: 1. Early presbyopia. 2. Primary closed angle glaucoma. 3. Convergent squint.		
Treatment	Treatment of the Cause: e.g. 1. Hyperglycemia. 2. Irido-cyclitis. Treatment of the Condition: ✎ 1. Spectacles lens: ✎ ➤ Characters: Spherical (bounded by 2 surfaces each is a portion of a sphere) – Concave (minus) to move the image backwards to the retina.	Treatment of the Cause: e.g. 1. Hypoglycemia. Treatment of the Condition: ✎ 1. Spectacles lens: ✎ ➤ Characters: Spherical (bounded by 2 surfaces each is a portion of a sphere) – Convex (plus) to move the image forward to the retina.	Treatment of the Cause: e.g. 1. Keratoconus: by Intra-corneal segments. 2. Lens subluxation: by lens removal and IOL implant. Treatment of the Condition: Regular Astigmatism ✎ 1. Spectacles lens: ✎ ➤ Characters: Cylindrical (cut from a cylinder in a plane parallel to its axis ⇒ rays of light passing in a plane parallel to the axis of the cylinder undergo no refraction while rays passing in a plane at right angle to the axis undergo maximum refraction).
	➤ Disadvantages (in high errors): ◎ Making change in retinal image size. ◎ Spherical aberration (i.e. straight lines appear curved) ◎ Poor coordination of manual movements (e.g. placing a key in a lock). ◎ Restricted V.F. ◎ Unsuitable in unilateral cases ⇒ anisokonia and binocular diplopia. ◎ Cosmetically unacceptable.		
	2. Contact lens: ✎ (N.B. both CL and refractive surgery are less tolerated and successful in hypermetropia than in myopia). ➤ Characters: the same as mentioned in spectacles lens (+ the CL used in regular astigmatism is Toric i.e. special soft CL). ➤ Advantages: the reverse of the advantages of spectacles lens. ➤ Disadvantages: ◎ Special care is required for its cleanliness and storage. ◎ Some people don't tolerate them. ◎ Infection is a risk with bad hygiene. ◎ Traumatic corneal abrasions may occur during manipulation. ◎ Some people develop allergy their related solutions.		
	3. Refractive surgery: ✎ (3 corneal – 2 intra-ocular) ➤ Radial keratotomy: ◎ Indication: low myopia. ◎ Principle: radial incisions were done to weaken the cornea and make it flatter by the effect of IOP.	3. Refractive surgery: ✎ (2 corneal – 1 intra-ocular)	3. Refractive surgery: ✎ (3 corneal) ➤ Astigmatic keratotomy: ◎ Principle: corneal incisions are done to flatten the steep meridian.
	➤ Photorefractive Keratectomy (PRK): ◎ Indication: moderate myopia, low hypermetropia and astigmatism. ◎ Principle: excimer laser ablation is done on the surface of the cornea after removing the overlying epithelium. ➤ Laser In-situ Kerato-mileusis (LASIK): ◎ Indication: moderate myopia, low hypermetropia and astigmatism. ◎ Principle: ✎ steps: ✎ Corneal flap is made using a special microkeratome – ✎ Excimer laser is directed to the center of the cornea ⇒ removing part of the corneal thickness in a calculated manner – ✎ The flap is returned again.		
	➤ Clear lens extraction ➤ Phakic IOL (high myopia) Treatment of the Complications: e.g. squint.	➤ Phakic IOL: always discuss IOL as in aphakia ☺ Treatment of the Complications: e.g. squint.	Irregular Astigmatism ✎ 1. CL: rigid (to make irregular surface ⇒ regular one) 2. Penetrating keratoplasty.

Aphakia	Anisometrpoia	Presbyopia																				
Definition: ✂ Absence of the crystalline lens in the pupillary area.	Definition: ✂ Significant difference in refraction between the two eyes (more than 4 D).	Definition: ✂ Recession of the near point, due to progressive weakness of the accommodation with aging ⇒ uncomfortable near vision																				
Etiology: ✎ ✂ Surgical removal. ✂ Posterior lens dislocation (e.g. due to trauma or hyper-mature cataract). ✂ Congenital (very rare).	Types: ✂ <u>Simple</u> : one eye is emmetropic and the other is ametropic. ✂ <u>Compound</u> : both eye are ametropic but of the same type. ✂ <u>Mixed</u> : one eye is myopic and the other is hyper-metropic.	Etiology: ✂ With aging ⇒ the lens becomes hard (loses its ability to change its dioptric power) and ciliary muscles becomes weak.																				
Symptoms and complications: ✎ ✂ Shift of refractive state to Hypermetropia. ✂ Secondary glaucoma (due to pupillary block by the herniating vitreous). ✂ Irido-cyclitis.	Clinical picture and complications: ✂ Asthenopia (e.g. burning sensation, etc.). ✂ Binocular diplopia. ✂ Amblyopia. ✂ Squint (convergent and divergent according to the age and type of causative).	Symptoms: ✂ <u>Difficult near vision:</u> ☁ If the patient was previously emmetropic: this occurs around the age of 40 ☁ If the patient was previously myopic: this occurs later. ☁ If the patient was previously hyper-metropic: this occurs earlier.																				
Signs: ✎✎✎ ✂ <u>Anterior chamber:</u> Deep. ✂ <u>Iris:</u> tremulous. ✂ <u>Pupil:</u> jet black. ✂ If the cause is surgical removal: scar is found. ✂ If the cause is posterior dislocation: lens can be seen by fundus exam. ✂ 1 <u>Purkinje-Sanson image</u> is present. 👁 <u>N.B.</u> <u>Purkinje-Sanson images:</u> <table><tr><th>Image</th><th>Formed by</th><th>Size</th><th>Position</th><th>Movement</th></tr><tr><td>1</td><td>Cornea</td><td>Small</td><td>Erect</td><td>With</td></tr><tr><td>2</td><td>Anterior lens capsule</td><td>Small</td><td>Erect</td><td>With</td></tr><tr><td>3</td><td>Posterior lens capsule</td><td>Very small</td><td>Inverted</td><td>Against</td></tr></table>	Image	Formed by	Size	Position	Movement	1	Cornea	Small	Erect	With	2	Anterior lens capsule	Small	Erect	With	3	Posterior lens capsule	Very small	Inverted	Against	Treatment: ✂ <u>Treatment of the Condition:</u> ✎ 1. <u>Spectacles lens</u> (the same as mentioned before but mainly anisokonia i.e. un-equal retinal images). 👁 <u>N.B.</u> <u>Spectacles can be only used</u> after under-correcting the eye with a higher error. 2. <u>CL</u> . (reduces difference of retinal images to 6%) ⦿ Advantages and disadvantages: as before. 3. <u>Refractive surgery:</u> the best choice making the difference negligible ✂ <u>Treatment of the Complications:</u> e.g. Amblyopia and squint.	<u>Accommodative asthenopia</u> (e.g. burning sensation, etc.). Treatment: ✂ <u>Treatment of the Condition:</u> ✎ 1. <u>Spectacles lens</u> ⦿ Characters of used lens: convex i.e. plus (added to the far correction) to compensate for the lost automatic focusing power of the lens. 2. <u>CL</u> . ⦿ Characters of used lens: convex - multifocal (limited success). 3. <u>Refractive surgery:</u> multifocal Intra Ocular Lenses implantation.
Image	Formed by	Size	Position	Movement																		
1	Cornea	Small	Erect	With																		
2	Anterior lens capsule	Small	Erect	With																		
3	Posterior lens capsule	Very small	Inverted	Against																		
Treatment: ✂ <u>Treatment of the Cause:</u> e.g. managing the other effects of trauma. ✂ <u>Treatment of the Condition:</u> ✎ 1. <u>Spectacles lens</u> . 2. <u>CL</u> . (both are the same as hypermetropia). 3. <u>Refractive surgery:</u> 1 <u>intra-ocular</u> ⇒ <u>Aphakic IOL</u> (= <u>psudo-phakia</u>) ⦿ Characters of used lens: as spectacles and CL (i.e. spherical convex). ⦿ Sites of implantation: sulcus, angle, iris or bag supported (AC or PC). ⦿ <u>Materials of used lens:</u> Rigid (PMMA-made) or Foldable (silicon-made). ⦿ Timing: 1 ^{ry} (at the time of removing original lens) or 2 ^{ry} (later on). 👁 <u>N.B.</u> if the patient is younger than 1 year ⇒ spectacles then 2 ^{ry} IOL is implanted. ✂ <u>Treatment of the Complications:</u> e.g. Irido-cyclitis.	Symptoms (of asthenopia): burning, headache, hyperemia, dryness ⇒ blinking and lacrimation. Causes (of asthenopia): ✂ <u>Accommodative:</u> Hypermetropia, Astigmatism, Presbyopia and Anisometrpoia. ✂ <u>Muscular:</u> myopia and Latent squint (due to disproportion () convergence and accommodation).	Miscellaneous Indications of CL and Refractive surgery: ✂ <u>Optical:</u> high refractive errors ✂ <u>Mechanical:</u> e.g. nose disfigurement. ✂ <u>Cosmetic:</u> e.g. in people working in media. ✂ <u>Therapeutic (in CL):</u> to treat corneal disease.																				
Asthenopia																						
Definition: group of symptoms noticed with visual tasks chiefly after close work, especially in the evening by artificial illumination.																						

lens		
	Congenital (Developmental) cataract	Senile cataract
Definition	👁️ Opacification of the crystalline lens discovered at birth or in the early years of life.	👁️ Bilateral progressive opacification of the crystalline lens affecting old people above 50 years as an ageing process.
Etiology	👁️ <u>Metabolic disorders</u> e.g. galactosemia. 👁️ <u>Intra-uterine infections</u> especially rubella "German measles". 👁️ <u>Hereditary</u> . 👁️ <u>Mal-nutrition</u> e.g. Iron and calcium deficiency.	👁️ <u>Unknown</u> , however it may be due some changes with old age: ➤ Disturbed lens metabolism (↓ed oxygen uptake and ↑ed water content). ➤ Prolonged exposure to ultraviolet rays. ➤ Endocrinal disturbances due to age. ➤ Disturbance of permeability of the capsule. 💀 <u>N.B.</u> The lens which is affected by these changes undergoes hydration, dehydration or coagulation of its proteins.
Symptoms	👁️ White pupil (leukocoria). 👁️ Decreased vision. 💀 <u>N.B.</u> Symptoms are usually told by the mother.	👁️ Gradual painless progressive <u>diminution of vision</u> . 👁️ Progressive <u>myopia</u> (due to increased refractive index of the nucleus). 👁️ <u>Impaired night vision</u> : in cortical cataract. 👁️ <u>Impaired day vision</u> : in nuclear cataract. 👁️ <u>Fixed black spots</u> in the visual field. 👁️ <u>Colored halos around light</u> (due to fluid droplets inside the lens in early stages). 👁️ <u>Uni-ocular diplopia or polyopia</u> (due to irregular refraction by scattered lens opacities in immature cataract).
Signs and Invest.	📖 Local lens examination (Morphological types) 🙅🙅🙅 <u>1. Anterior polar cataract:</u> 👁️ <u>Cause:</u> delayed formation of AC in intra-uterine life ⇒ long contact between the lens and the cornea. 👁️ <u>Shape:</u> pyramidal, reduplicated, hour glass or collar stud. 👁️ <u>Effect on vision:</u> mild. <u>2. Posterior polar cataract:</u> 👁️ <u>Cause:</u> remnants of the hyaloid artery. 👁️ <u>Shape:</u> small disc shaped opacity at the posterior pole. 👁️ <u>Effect on vision:</u> severe as the opacity is near the nodal point of the eye. <u>3. Lamellar (Zonular) Cataract:</u> 👁️ <u>Cause:</u> mal-nutrition ⇒ opacification involving 1 or more layers of lens but the rest of the layers are clear 👁️ <u>Shape:</u> central white disc with spokes like a steering wheel of a ship. 👁️ <u>Effect on vision:</u> variable.	📖 Local lens examination (Morphological types) 🙅 + 🙅 👁️ <u>Nuclear cataract:</u> in which opacification affects mainly the nucleus in 2 stages: <u>1. Stage of sclerosis:</u> physiological aging process in which there is gradual ↓ of water content (dehydration) and ↑ in refractive index ⇒ index myopia. 💀 <u>N.B.</u> RR in this stage appears normal (all red) and vision can be corrected by glasses. <u>2. Stage of opacification:</u> pathological process in which the lens loses its transparency and acquires a yellowish and later brownish or black color (due to deposition of melanin and tryptophan-derived pigments). 💀 <u>N.B.</u> RR appears black centrally with redness in periphery and vision can't be corrected by glasses (needs surgery). 👁️ <u>Cortical cataract:</u> in which opacification affects mainly the cortex in 3 stages: <u>1. Immature stage:</u> in which the lens is not totally opaque and divided into 2 sub-stages: 👁️ <u>Incipient sub-stage:</u> in which the lens is almost clear with the presence of some opaque fibers (so the vision is good ranging between 6/6 to 6/9). 💀 <u>N.B.</u> if the opaque fibers are anterior fibers ⇒ incipient cuneiform cataract, while if they are posterior fibers ⇒ incipient cupuliform cataract. 👁️ <u>Typical immature sub-stage:</u> in which the lens is almost opaque with the presence of some clear fibers under the anterior capsule (so the vision worsens). <u>2. Mature Stage:</u> in which the lens is totally opaque (so the vision drops to HM). <u>3. Hyper-mature stage:</u> divided into 2 forms (types): 👁️ <u>Wet type (Morgagnian cataract):</u> in which the cortex becomes soft, liquefied and milky ⇒ sinking of the nucleus the bottom of the capsule.

Signs and Invest.	<u>4. Total Cataract:</u> ✎ <u>Cause:</u> rubella infection to the mother in the first trimester . ✎ <u>Shape:</u> take the shape of the whole lens. ✎ <u>Effect on vision:</u> very severe. ⚠ N.B. It is usually associated with heart anomalies and other congenital anomalies. <u>5. Punctate or Blue-Dot Cataract:</u> multiple small bluish dots. <u>6. Fusiform Cataract (coralliform):</u> antero-posterior spindle shaped opacity. <u>7. Disciform Cataract.</u> <u>8. Sutural:</u> either anterior or posterior. <u>9. Coronary Cataract:</u> ring of pear-shaped or oval opacities with their apices pointing peripherally seen in the cortex near the equator. ⚠ N.B. Punctate, Fusiform, Disciform, Sutural and Coronary cataract usually have little or no effect on vision. 📖 Local examination of the rest of the eye: e.g. ✎ <u>Fundus examination:</u> to exclude retinal anomalies which can also cause white pupil e.g. retinoblastoma. ✎ <u>Visual assessment:</u> by preferential looking test or visual evoked potentials (VEP). 📖 General examination and Investigations: e.g. ✎ <u>Laboratory tests:</u> to exclude metabolic causes when suspected e.g. galactosemia.	✎ <u>Dry type:</u> in which: ✎ Size of the lens: shrunken due to loss of water. ✎ AC: Deep with tremulous iris. ✎ Capsule: thickened, wrinkled with calcium depositions. ✎ Tension: ↑ed (Phacolytic 2ry open-angle glaucoma) as escaping lens proteins in the aqueous excite a phagocytic response and then swollen macrophages may get trapped in the trabecular meshwork. <u>4. Intumescent cataract:</u> special type occurring in some cases in which: ✎ Size of lens: swollen because it draws water from the aqueous. ✎ AC: shallow. ✎ Capsule: glistening. ✎ Tension: ↑ed (Phaco-morphic 2ry closed-angle glaucoma) due to block of the pupil by the swollen lens. <table border="1" data-bbox="1130 600 2792 1125"> <thead> <tr> <th colspan="3">N.B. METHODS HELPING IN CATARACT EXAMINATION</th></tr> <tr> <th></th><th>Iris shadow</th><th>Red reflex (more reliable)</th></tr> </thead> <tbody> <tr> <td>Definition</td><td>shadow of the iris on the lens opacity (when the light is thrown obliquely "45°" on it).</td><td>red reflection of light from retina in dark room.</td></tr> <tr> <td>To be formed</td><td>clear interval between the iris and the opacity is needed</td><td>clear lens (totally or partially) is needed.</td></tr> <tr> <td>Normally</td><td>not present as no opacity</td><td>present as no opacity.</td></tr> <tr> <td>Present in</td><td>immature and hyper-mature cataracts (clear interval here is aqueous as lens shrunk)</td><td>normal eye (total), nuclear and immature cortical cataracts (partial).</td></tr> <tr> <td>Absent in</td><td>mature and intumescent cataracts</td><td>mature, hyper-mature and intumescent cataracts</td></tr> </tbody> </table> 📖 Local examination of the rest of the eye: usually free e.g. ✎ <u>Visual acuity.</u> ✎ <u>Lacrimal sac examination:</u> by regurgitation test ✎ <u>Eyelids examination:</u> for rubbing lashes, blepharitis, styne or chalazion. ✎ <u>Conjunctival swab.</u> ✎ <u>Slit-lamp examination:</u> to assure: ➤ No signs of irido-cyclitis. ➤ Well reactive pupil (good test for optic nerve function). ✎ <u>Fundus examination:</u> by ophthalmoscope to diagnose any retinal or optic nerve disease. ✎ <u>Retinal function tests:</u> "when the fundus can't be seen as in mature cataract" ➤ Light projection to test retinal periphery. ➤ Color perception to test macula. ✎ <u>Ultrasonography.</u> 📖 General examination and Investigations: usually free e.g. ✎ CVS examination. ✎ Chest examination. ✎ Urine analysis: for sugar, pus cells and albumin. ✎ Search for septic foci: in teeth, ear, nose and throat.	N.B. METHODS HELPING IN CATARACT EXAMINATION				Iris shadow	Red reflex (more reliable)	Definition	shadow of the iris on the lens opacity (when the light is thrown obliquely "45°" on it).	red reflection of light from retina in dark room.	To be formed	clear interval between the iris and the opacity is needed	clear lens (totally or partially) is needed.	Normally	not present as no opacity	present as no opacity.	Present in	immature and hyper-mature cataracts (clear interval here is aqueous as lens shrunk)	normal eye (total), nuclear and immature cortical cataracts (partial).	Absent in	mature and intumescent cataracts	mature, hyper-mature and intumescent cataracts
N.B. METHODS HELPING IN CATARACT EXAMINATION																							
	Iris shadow	Red reflex (more reliable)																					
Definition	shadow of the iris on the lens opacity (when the light is thrown obliquely "45°" on it).	red reflection of light from retina in dark room.																					
To be formed	clear interval between the iris and the opacity is needed	clear lens (totally or partially) is needed.																					
Normally	not present as no opacity	present as no opacity.																					
Present in	immature and hyper-mature cataracts (clear interval here is aqueous as lens shrunk)	normal eye (total), nuclear and immature cortical cataracts (partial).																					
Absent in	mature and intumescent cataracts	mature, hyper-mature and intumescent cataracts																					
Comp.	✎ <u>Amblyopia and squint:</u> in unilateral cases. ✎ <u>Nystagmus:</u> in bilateral cases.	✎ Phacolytic glaucoma. ✎ Iritis or Endophthalmitis Phaco-anaphylactica. ✎ Phaco-toxic uveitis. ✎ Subluxation or dislocation of the lens																					

Treatment	Prophylaxis and treatment of the cause: e.g. malnutrition.			Treatment of the Condition: surgical treatment		
	Treatment of the Condition: surgical treatment			1. Factors ruling the surgical treatment: individual needs of each patient.		
	1. Factors ruling the surgical treatment:			2. Surgical techniques:		
	➤ Before 3 years: density of cataract (seen by ophthalmoscope). ✂ If you cannot see the fundus details through a dilated pupil: do surgery. ➤ After 3 years: visual acuity test. ✂ If vision is 6/18 or more: no need for surgery (because this amount of vision with accommodation is better than good visual acuity without it). ✂ If vision is< 6/18: do surgery early.			➤ <u>Extra-capsular cataract extraction (ECCE)</u>: standard technique ✂ Principle: Removing lens leaving the posterior capsule intact ⇒ New IOL can be implanted in P.C or A.C. and Iridectomy is not indicated. ✂ Methods of hard nucleus removal: ✂ Delivery through a large incision (10-12 mm) at the cornea or the limbus. ✂ Phaco-emulsification (using a special ultrasound machine) then aspiration through a small incision (3mm) at the cornea or the limbus.		
	💀 <u>N.B.</u> Early restoration of vision is important to avoid amblyopia and nystagmus.			➤ <u>Intra-capsular cataract extraction (ICCE)</u> : rarely used now		
	2. Surgical techniques:			✂ Principle: Removing the lens within its capsule after zonulysis ⇒ New IOL should be implanted in A.C. and Iridectomy is indicated (to prevent pupillary block)		
	➤ Irrigation and Aspiration of lens matter through anterior segment, followed by anterior vitrectomy. ➤ Pars plana Lensectomy and vitrectomy. (using vitrectomy probe).			✂ Dangers: vitreous loss, retinal detachment and macular edema		
3. Visual rehabilitation: to correct aphakia (discuss from errors 😊)			3. Visual rehabilitation: to correct aphakia (discuss from errors 😊)			
Treatment of the Complications: e.g. amblyopia.			Treatment of the Complications: e.g. glaucoma and uveitis.			
	Senile Nuclear cataract	Senile cortical cataract				
		Immature	Mature	Hyper-mature (dry)	Intumescent	
Vision	↓ed duo to index myopia	6/6 : 6/9 in incipient sub-stage	HM		CF : HM	
		6/9 : CF in typical sub-stage				
Shape	Described before in details					
Size	Normal size lens			Shrunken lens	Swollen lens	
AC	Normal			Deep with tremulous iris	shallow	
Capsule	Normal			Thickened, wrinkled with calcium depositions	Glistening	
Iris shadow	Present		Absent	Present	Absent	
RR	Partially present		Absent			
Tension	Normal			Phacolytic glaucoma	Phaco-morphic glaucoma	
Specific	<u>Index myopia</u>	<u>Uni-ocular diplopia or polyopia</u>	-	Complications of hyper (write)	Phaco-morphic glaucoma	

	Complicated cataract	Traumatic cataract
Definition	Opacification of the crystalline lens due to systemic or ocular causes.	Opacification of the crystalline lens due to ocular trauma.
Etiology	📖 Systemic causes: e.g. 1. Metabolic: 👉 🤝 👉 Diabetes Mellitus: 2 types may occur: 👉 True diabetic cataract (Snow flake cataract): ill-defined punctate opacities which mature rapidly if DM is not controlled in young patients. 👉 Pre-senile cataract: similar to senile cataract but at a younger age. 👉 Galactosemia: metabolic defect of galactose mechanism ⇒ cataract in the first life month (preventable). 👉 Hypoparathyroidism. 👉 Hypocalcaemia. 2. Toxic medications: 3. Exposure to radiant: 4. Skin diseases: 👉 Systemic steroids. 👉 Infrared rays, microwaves and 👉 Atopic dermatitis. 👉 Naphthalene and Thallium. high radiation. 👉 Ectodermal dysplasia. 👉 Ergot. 👉 X-ray and electric shock. 📖 Local (Ocular) causes: e.g. 1. Toxic medications: 2. Degenerative diseases: 3. Ocular inflammations: 👉 Local steroids. 👉 High myopia. 👉 Severe keratitis. 👉 Pilocarpine. 👉 Absolute glaucoma. 👉 Chronic irido-cyclitis. 👉 Adrenaline. 👉 Long-standing RD. 👉 Chronic chorio-retinitis. 👉 Retinitis pigmentosa. 👉 N.B. These causes interfere with lens metabolism ⇒ opacification of lens fibers.	👉 Blunt trauma. 👉 Perforating trauma. 👉 N.B. the opacification induced by perforating trauma (i.e. with ruptured capsule) can be also called "after-cataract" 👉 Intra-ocular foreign body e.g. iron and copper
Symptoms	Usually symptoms of the underlying cause.	
Signs and Invest.	📖 Local lens examination (Morphology) 👉 Site: posterior cortex at the beginning (as the posterior capsule is thin and devoid of sub-capsular epithelium) then later on, whole lens is affected. 👉 Shape and Color: grey rosette shaped cataract with poly chromatic luster at the beginning, then later on, chalky white cataract. 📖 Local examination of the rest of the eye: the same as senile but here it shows the underlying cause e.g. 👉 Visual acuity: doesn't depend on the cataract density only but also on underlying ocular disease (e.g. HM vision may be found with relatively clear lens) 👉 Slit-lamp examination: shows signs of irido-cyclitis e.g. KPs may be found. 📖 General examination and Investigations: the same as senile but it shows the underlying systemic cause e.g. 👉 CVS examination. 👉 Chest examination. 👉 Urine analysis: for sugar, pus cells and albumin. 👉 Search for septic foci: in teeth, ear, nose and throat.	📖 Local lens examination (Morphology) 1. Blunt trauma (concussion) cataract: 👉 Site: posterior cortex(due to post. capsule rupture). 👉 Shape: rosette shaped cataract. 👉 Associated with: Vossius ring (circle of iris pigment on the anterior lens capsule due to the impress of the pupillary border of the iris on the lens) 2. Perforating trauma cataract: or "after cataract" 👉 Shape: localized opacity or total cataract (depending on the size of the anterior capsule injury). 3. Intra-ocular F.B. cataract: according to the FB e.g. 👉 If the FB is iron: siderotic cataract. 👉 If the FB is copper: sunflower cataract.

Signs and Invest.	📖 Local examination of the rest of the eye in traumatic cataract: <i>the same as the complicated but here it shows the other possible effects of the trauma.</i>		
	📖 General examination and Investigations in traumatic cataract: <i>not necessary if the trauma is localized to the eye.</i>		
Treatment	🔪 Prophylaxis and treatment of the cause: ➤ by well caring and treatment of possible ocular and systemic causes.		🔪 Treatment of the Cause: <i>i.e. dealing with trauma</i> ➤ Removing Intra-ocular foreign body. ➤ Proper suturing of wounds. ➤ Regular follow up (to detect any late complications of the trauma).
	🔪 Treatment of the Condition: <i>surgical treatment</i> 🧑🏻		
	1. Factors ruling the surgical treatment: the state of the eye apart from the cataract (decreased vision should be due to cataract. otherwise, cataract extraction will not be beneficial to the patient).		
	2. Surgical techniques and: <i>depends on the age:</i> ➤ Soft cataract (i.e. less than 25 years old): the same as congenital cataract. ➤ Hard cataract (i.e. more than 25 years old): the same as senile cataract.		
	3. Visual rehabilitation: to correct aphakia (discuss from errors 😊)		
	After cataract		
Definition	🔪 Opacified lens matter in the pupillary area following cataract operation or trauma. 💀N.B. Cataract is opacified lens with intact capsule while after cataract is opacified lens with open capsule i.e. by surgery or trauma.		
Etiology	🔪 Lens fibers were left behind during surgery. 🔪 Posterior capsule opacification. 🔪 Anterior sub-capsular epithelium was left during surgery or irritated by trauma ⇒ proliferation.		
Treatment	🔪 If vision is not affected: no interference is necessary. 🔪 If vision is affected: surgical removal or YAG-laser capsulotomy.		
	Lens subluxation	Anterior lens dislocation	Posterior lens dislocation
Definition	Displacement of the lens to one side due to tearing of a portion of the suspensory ligament (zonule).	Anterior displacement of the lens from its place due to total tearing or absence of the zonules.	Posterior displacement of the lens from its place due to total tearing or absence of the zonules.
Etiology	🔪 Metabolic disorders e.g. homocystinuria. 🔪 Ocular Trauma. 🔪 Degenerative disorders e.g. hyper-mature cataract. 🔪 Congenital disorders e.g. Marfan's syndrome.		
Clinical picture and complications	🔪 Myopic astigmatism. 🔪 Uni-ocular diplopia. 🔪 Irregular depth AC. 🔪 Lens edge may be seen in pupil.	🔪 Myopia. 🔪 Corneal decompensation and edema. 🔪 Lens is like oil globule in AC.	🔪 Hypermetropia. 🔪 Deep AC. 🔪 Other signs of aphakia. 🔪 Lens is seen by fundus examination.
	🔪 Tremulous iris.	🔪 Irido-cyclitis.	🔪 Tremulous iris. 🔪 Irido-cyclitis.
Complications	🔪 Secondary glaucoma (Pupillary block glaucoma or glaucoma inversus) that worsens with miotics and is relieved by mydriatics. 💀N.B. Pupil is blocked by the lens or the herniating vitreous in the posterior dislocation. 💀N.B. Posterior dislocation can also cause Phaco-anaphylactic glaucoma.		
Treatment	🔪 Treatment of the cause. 🔪 Treatment of the complications.		
	🔪 Treatment of condition: ☁ If not complicated: glasses or CL ☁ If severely complicated: Removal.	🔪 Treatment of condition: ☁ Removal through a corneal incision.	🔪 Treatment of condition: ☁ Removal by pars plana vitrectomy.

	Glaucoma	
	Primary open angle glaucoma	Primary closed angle glaucoma
Definition	✎ Bilateral Asymmetrical progressive genetically determined disease in which: ☼ IOP is elevated $\Rightarrow$ visual field defects and optic disc changes. ☼ Angle of the AC is wide when viewed by Gonioscopy.	✎ Acute attacks of IOP elevation due to sudden closure of the A.C angle which: ☼ Occurs after exposure to precipitating factors in patients with risk factors. ☼ May be preceded by mild intermittent (prodromal) attacks. ☼ If not well treated $\Rightarrow$ chronicity with visual field and optic disc affection.
Etiology	✎ Risk factors: ☼ Old age > 45 years-old. ☼ Dark races. ☼ Strong family history. ☼ Ocular hypertension. ☼ Myopia (genetically linked). ☼ Vasospastic disorders e.g. migraine. ✎ Cause: idiopathic trabecular sclerosis.	✎ Risk factors: ☼ Essential: small eye with crowded structures (i.e. shallow AC, narrow angle and strong contact between the iris and the lens). ☼ Additional: $\Rightarrow$ old-aged nervous females > 40 years-old (menopausal). ✎ Cause (precipitating factors): pupillary dilatation (mydriasis) e.g. from emotional excitement, dilating drops or staying in the dark.
Symptoms	✎ Asymptomatic (so it may steal vision if not early discovered). ✎ Night blindness (defective dark adaptations). ✎ Visual field defects.	1. Intermittent (Prodromal) stage: Symptoms: Transient attacks of: ✎ Ocular pain (mild) and headache. ✎ blurring of vision. ✎ Colored haloes. Signs: Transient attacks of: ✎ IOP elevation. ✎ Small eye (☺). ✎ Dilated pupil. ✎ Corneal edema (mild). ⚠ N.B.₁ The attack may be broken after 1-2h by physiological miosis (e.g. light or sleep). ⚠ N.B.₂ The eye may look normal in between the attacks (no symptoms and little signs)
Signs	1. Elevation of IOP: ☼ Measured by: tonometers (indentation – applanation – air-puff non-contact). ☼ Values: $\Rightarrow$ normal = 10-22 mm Hg. $\Rightarrow$ Suspicious = 22-26 mm Hg. $\Rightarrow$ Diagnostic > 26 mm Hg. ⚠ N.B. Difference between 2 eyes and diurnal variation shouldn't be > 4 mm Hg. 2. Visual field defects: ☼ Measured by: perimetry ☼ Factors affecting: distribution of NFL as they converge toward the OD e.g. $\Rightarrow$ The most crowded fibers: arcuate fibers (temporal fibers above and below the macula) $\Rightarrow$ the earliest V.F. defects occur in this zone. $\Rightarrow$ The least crowded fibers: macular fibers $\Rightarrow$ lately affected with V.A sparing $\Rightarrow$ Same horizontal distribution of nerve fibers at the retina and OD. ☼ They include: $\Rightarrow$ Enlargement of blind spot. $\Rightarrow$ Paracentral scotoma: isolated in the area between 10-20° of fixation. $\Rightarrow$ Upper or lower arcuate scotoma (Bjerrum's scotoma): formed when paracentral scotoma elongates circumferentially along the distribution of arcuate nerve fibers to connect with the blind spot. $\Rightarrow$ Double arcuate scotoma (Ring scotoma): When both upper and lower arcuate scotoma are both formed. ⚠ N.B. Ring scotoma is horizontally asymmet. $\Rightarrow$ nasal Roenne step or temporal wedge. $\Rightarrow$ Peripheral concentric contraction of the field (Breakthrough). $\Rightarrow$ Tubular vision (small area of central vision) with temporal island. $\Rightarrow$ Temporal island: the last remaining area. ⚠ N.B. Difference between 2 eyes and diurnal variation shouldn't be > 4 mm Hg.	Investigations: Provocative tests (done when you suspect and want to prove) ✎ Principle: measuring IOP before and after exposing the eye to mild precipitating factors $\Rightarrow$ rise of IOP by 8 mmHg or more is diagnostic. ✎ Methods: ☼ Mydriatic test (by mild Mydriatic eye drops e.g. Phenylephrine 5%). ☼ Dark room test (by allowing the patient to stay awake in the dark for 1hour). Treatment: Provocative tests (done when you suspect and want to prove) ✎ Prophylaxis: ☼ Prevent people with RF from exposure to precipitating factors. ☼ Preventing further attacks by miotics, peripheral iridectomy or laser iridotomy. 2. Acute stage (Acute congestive glaucoma): Symptoms: (as intermittent but severe – haloes + NV and PBL) ✎ Ocular pain (severe bursting) (due to stretch of ocular coats) and headache (severe). $\Rightarrow$ ✎ Nausea and vomiting (due to reflex vagal stimulation). ✎ Photophobia, blepharo-spasm and lacrimation. ✎ Sudden $\downarrow$ed vision (severe). Signs: (as intermittent but severe + fundus) ✎ IOP elevation (severe up to 70 mmHg i.e. stony hard eye by digital palpation). ✎ Small eye (☺). ✎ Semi-dilated irreactive vertically oval pupil. ✎ Corneal edema, haze (severe), Eyelid edema and Ciliary congestion. ✎ Fundus is hardly seen (due to corneal edema) ✎ V.A: down to PL.

Signs	3. Optic disc changes (Cupping): ☼ <u>Examined by:</u> ophthalmoscopy (fundus examination). ☼ <u>Early changes:</u> ➤ Large cup/disc ratio (0.4 - 0.7). (⚠ N.B. normal ratio is 0.3). ➤ Asymmetry of C/D ratio between the 2 eyes. ➤ Vertical elongation of the optic cup. ➤ Notching of the rim of the cup at the upper and lower pole. ➤ Retinal nerve fiber layer defects (seen with green filter). ➤ Splinter hemorrhage on the disc. ➤ Increased visibility of the pores of the lamina cribrosa. ☼ <u>Late changes:</u> ➤ Large cup/disc ratio (more than 0.7). ➤ Deep cup with undermined edges (billiard table). ➤ Nasal shift of the vessels.	Fate: ✗ Recovery: either with treatment or spontaneous (very rare due to miosis when the patient sleeps or relieve of pupillary block due to iris atrophy). ✗ Turn to chronic stage: with formation of PAS. ✗ Turn to absolute stage: with complete loss of vision in severe attack. Treatment: ☉ <u>Prophylaxis:</u> ✗ Prevent people with RF from exposure to precipitating factors. ✗ Preventing further attacks (in the other eye) by miotics, peripheral iridectomy or laser iridotomy. ☉ <u>Treatment of the condition:</u> essentially surgical ✗ <u>Pre-operative medications:</u> ➤ Aim: to ⇒ the very high IOP prior to surgery and Relieve the patient's suffering ➤ Include: hospitalization and: ◆ Topical Miotics e.g. Pilocarpine 2-4% every 30 minutes until the pupil constricts. ◆ Analgesics and Anti-emetics. ◆ Hyperosmotic agents e.g. Mannitol 20-25% solution intravenous, 1-2 mg/Kg body . weight, 60 drops / minute to elevate the plasma osmotic pressure ⇒ withdrawing fluids from the eye into the plasma.
Investigations	✗ <u>Optical Coherence Tomography (OCT):</u> ☼ <u>Description:</u> sophisticated computer dependent machine that uses plane-polarized light to measure the thickness of the NFL around the optic nerve head. ☼ <u>Value:</u> early diagnosis and follow up as early damage signs appears in NFL.	
Treatment	✗ <u>Prophylaxis:</u> ☼ Early diagnosis by routine checking IOP in people with RF (enumerate). ☼ Follow up by routine V.F examination in patients to prevent more damage. ✗ <u>Condition:</u> ⚠ 1. Medical treatment: main line 🖐 ➤ Miotics: ☉ <u>Route and examples:</u> topical Pilocarpine nitrate 1-4 %. ☉ <u>Mechanism:</u> ↑ aqueous trabecular outflow. ☉ <u>Dangers:</u> pupillary block glaucoma, cataract, ↓ vision in nuclear cataract, V.F constriction and spasm of accommodation – GIT colic and headache. ➤ Adrenergic Agonists: ☉ <u>Route and examples:</u> topical Epinephrine and Brimonidine ☉ <u>Mechanism:</u> ↑ aqueous trabecular outflow. ➤ Prostaglandin analogues: ☉ <u>Route and examples:</u> topical Xalatan, Travatan, Rescula or Lumigan ☉ <u>Mechanism:</u> ↑ aqueous uveo-scleral outflow.	◆ Oral or IV CAIs e.g. Acetazolamide to lower the IOP before surgery. ✗ <u>Surgical technique:</u> ➤ If the acute attack is recent (within 24 hours) and Gonioscopy shows no evidence of PAS ⇒ iridectomy is indicated. ➤ If the attack has lasted longer and Gonioscopy shows evidence of PAS ⇒ an external Fistulizing operation is performed (Sub-scleral trabeculectomy) in which connection is made between the AC and the sub-conjunctival space under scleral flap. 3. Chronic stage: Clinical picture: occurs if the acute stage is not properly managed with formation of PAS ✗ IOP is moderately elevated. ✗ Very shallow AC. ✗ Chronic corneal edema which may lead to bullous and filamentary keratopathy with less ciliary congestion. ✗ Semi dilated very sluggish pupil. ✗ Visual field defects.

Treatment	➤ Beta-blockers: ☹ <u>Route and examples:</u> topical Timolol (non-selective) or Betaxolol (selective) ☹ <u>Mechanism:</u> ↓ aqueous secretion. ☹ <u>Side effects:</u> broncho-spasm and bradycardia. ➤ Carbonic Anhydrase inhibitors (CAIs): ☹ <u>Route and examples:</u> topical Dorzolamide or systemic Acetazolamide. ☹ <u>Mechanism:</u> ↓ aqueous secretion. ☹ <u>Side effects:</u> allergic conjunctivitis – GIT, urinary, nervous affection. ⚠ <u>N.B.</u>₁ systemic CAIs are very effective but used only post-operative or in 2^{ry} cases. ⚠ <u>N.B.</u>₂ Medical treatment is the main line except if there's Progressive damage with maximum tolerated doses, Significant SE or/ Non-compliance of the patient. <u>2. Laser treatment (Laser trabeculo-plasty):</u> ➤ Type of laser: Argon or Double Frequency YAG ➤ Principle: low power laser burns ⇒ shrinkage and contraction of the trabecular ring ⇒ widening of the inter-trabecular spaces ⇒ help to drain aqueous. <u>3. Surgical treatment:</u> ✎ ➤ Sub-Scleral Trabeculectomy (SST): external Fistulizing operation communicating AC the sub-tenon or sub-conjunctival space ⇒ ↑aqueous drainage. ⚠ <u>N.B.</u> SST is done under a scleral flap to ↓ excessive filtration and severe Hypotony. ➤ Glaucoma valve (Device): large reservoir implanted between two recti muscles and connected to the AC with a silicon tube ⇒ ↑aqueous drainage. ➤ Ciliary body ablation: destruction of C.B epithelium ⇒ ↓aqueous secretion. ⚠ <u>N.B.</u> C.B. ablation is indicated in difficult cases unresponsive to other measures and can be done through Cyclo-diathermy, Cyclo-cryotherapy or Cyclo-photocoagulation.	Fate: ✎ Improvement and restoration of some vision: if well treated. ✎ Turn to absolute stage: with complete loss of vision if not treated. Treatment: ⊙ <u>Prophylaxis:</u> ✎ Well management of the acute stage. ⊙ <u>Treatment of the condition:</u> ✎ external Fistulizing operation is performed (Sub-scleral trabeculectomy) in which connection is made between the AC and the sub-conjunctival space under scleral flap. 3. Absolute stage: Symptoms: <i>end stage of uncontrolled glaucoma with high IOP.</i> ✎ Blind (no PL) painful eye. Signs: ✎ IOP is stony hard. ✎ Very shallow AC. ✎ Dilated, fixed and greenish-blue pupil. ✎ Chronic corneal edema and loss of sensations. ✎ Fundus shows optic atrophy. ✎ Iris shows atrophic patches. Sequelae: ✎ Complicated cataract. ✎ Scleral staphyloma (intercalary, ciliary and equatorial). ✎ Degenerative pannus: granulation tissue which invades the cornea at level of BM. ✎ Atrophia bulbi: due to ciliary body atrophy. Treatment: ⊙ <u>Prophylaxis:</u> ✎ Well management of the acute and chronic stages. ⊙ <u>Treatment of the condition:</u> <i>i.e. for blind painful eye</i> ✎ Ciliary body ablation e.g. cyclo-photocoagulation or Enucleation.
	Secondary open angle glaucoma	Secondary Closed angle glaucoma
Definition	✎ Elevation of the IOP secondary to local ocular disease (can be identified by the examination) with wide angle (viewed by the Gonioscopy) and normal depth AC.	✎ Elevation of the IOP secondary to local ocular disease (can be identified by the examination) with closed angle (viewed by the Gonioscopy) and shallow AC.
Causes	📖 <u>Inflammatory glaucoma: caused by:</u> 1. <u>Acute Irido-cyclitis</u> due to plasmoid aqueous and swelling of trabecular bores (treated by atropine, steroids for irido-cyclitis – BBs, CAIs for glaucoma). 2. <u>Herpes Simplex and zoster:</u> due to swelling of trabecular bores. 3. <u>Glaucomoto-cyclitic crisis:</u> attacks of unilateral rise of IOP mostly related	📖 <u>Inflammatory glaucoma: caused by:</u> 1. <u>Complicated and Chronic cases of Irido-cyclitis</u> due to synechia formation (treated surgically by Peripheral iridectomy or trabeculectomy). to disturbance of prostaglandin metabolism (usually with corneal edema - blurring)

Causes	📖 Neo-vascular glaucoma: 🔍 <u>Definition</u>: elevation of the IOP secondary to due to abnormal blood vessels growing from the iris surface to the angle of the anterior chamber, obstructing the trabecular meshwork and impairing aqueous outflow. 🔍 <u>Etiology</u>: CRVO (here it's also called 100-day glaucoma) – Diabetic retinopathy – Chronic irido-cyclitis. 🔍 <u>Clinical stages</u>: 🖐️ 1. <u>Stage of rubeosis iridis</u>: the abnormal vessels are limited to the surface of the iris. 2. <u>Stage of open angle glaucoma</u>: the abnormal vessels encroach on the angle, with subsequent leakage of proteins ⇒ Irido-cyclitis and subsequent OAG. 3. <u>Stage of open angle glaucoma</u>: The end stage where anterior synechiae closes the angle. 🔍 <u>Treatment</u>: 🖐️ ⦿ <u>Prophylaxis and treatment of the cause</u>: e.g. CRVO and DR. ⦿ <u>Treatment of the condition</u>: ➤ Treatment of the new vessels(early): Pan-retinal photocoagulation using argon or diode laser to cause regression of rubeosis and so control the rise of IOP. ➤ Treatment of the glaucoma: Glaucoma device or Ciliary body ablation (write them from POAG).	
	📖 Corticosteroid Induced Glaucoma: with prolonged use of steroids.	📖 Miscellaneous causes in iris: e.g. tumor or cysts or pushing the iris forwards by a rapidly growing posterior segment mass or intra ocular hemorrhage
	📖 Traumatic glaucoma: caused by: 1. <u>Traumatic acute irido-cyclitis</u> ⇒ inflammatory glaucoma ☺ 2. <u>Angle recession glaucoma</u>: due to tear of the ciliary body and recession of the angle structures backwards that may obstruct the aqueous outflow channels. 3. <u>Traumatic hyphema</u>: The blood in AC may obstruct the trabecular pores. 4. <u>Ghost cell glaucoma</u>: occurs in longstanding cases of vitreous hemorrhage where the RBCs breakdown and their rigid walls (ghost cells) diffuse into the aqueous and obstruct the trabecular pores.	📖 Traumatic glaucoma: caused by: 1. <u>Traumatic complicated irido-cyclitis</u> ⇒ inflammatory glaucoma ☺ 2. <u>Anterior lens dislocation</u>: due to pupillary block by the dislocated lens. 3. <u>Posterior lens dislocation</u>: due to pupillary block by the herniated vitreous and also Phaco-anaphylactic glaucoma.
	📖 Tumor-induced glaucoma: caused by: 🔍 The associated intra ocular inflammation. 🔍 Obstruction of the trabecular pores by the malignant cells.	📖 Tumor-induced glaucoma: caused by: 🔍 The associated intra ocular inflammation. 🔍 Obstruction of the epi-scleral veins by the growing tumor.
	📖 Lens-induced glaucoma: caused by: 🔍 <u>Phacolytic glaucoma</u>: ➤ <u>Mechanism</u> occurs in hyper-mature cataract where the lens proteins break down into small fragments that leak through the capsule into the AC where they are engulfed by the macrophages which are then trapped in the trabecular pores. ➤ <u>Treatment</u> lens extraction (by ECCE or ICCE) and correction of aphakia ☺ 🔍 <u>Phaco-anaphylactic glaucoma</u>: ➤ <u>Mechanism</u> follows trauma or surgery, where the lens proteins are released into the ocular fluid and initiate an autoimmune reaction, the products of which are deposited on the trabecular pores causing their narrowing and inflammation.	📖 Lens-induced glaucoma: caused by: 🔍 <u>Phaco-morphic glaucoma</u>: ➤ <u>Mechanism</u> occurs in intumescent cataract where the lens increases in size due to its high fluid content. It may encroach upon the anterior chamber, produce a pupillary block, or cause angle occlusion, resulting in closed-angle glaucoma. ➤ <u>Clinical picture</u> the same as acute congestive glaucoma with history of senile cataract. ➤ <u>Treatment</u> lens extraction (by ECCE or ICCE) and correction of aphakia ☺ 🔍 <u>Anterior and Posterior lens dislocation</u>: 🖐️ ➤ <u>Mechanism of glaucoma and Treatment</u>: see lens chapter ☺

Primary congenital glaucoma (Buphthalmos)		Differential diagnosis: <i>primary congenital glaucoma should be differentiated from:</i>	
Definition: ✎ Elevation of the IOP due to developmental anomalies at the drainage angle leading to stretching of the eye coats and enlargement of the globe.		1. <u>Other causes of large cornea:</u> <i>e.g.</i> ➤ Megalo-cornea: no corneal edema or Haab's striae, normal IOP, no optic cupping. ➤ High myopia. ➤ Kerato-globus: bilateral condition with familial incidence – occurs later in life with rupture of Descemet's membrane resulting in cloudy cornea.	
Incidence: ✎ 80% within the first 6 months of life. ✎ 80% in males. ✎ 80% bilateral. ✎ 10% autosomal recessive.		2. <u>Other causes of cloudy cornea at birth:</u> <i>e.g.</i> ➤ Birth trauma. ➤ Congenital rubella. ➤ Metabolic disorders.	
Etiology: <i>congenital anomaly at the angle which may be:</i> ➤ Abnormal mesodermal membrane at the anterior chamber angle. ➤ Failure of separation of the iris from the cornea. ➤ Abnormal insertion of ciliary muscles into trabecular meshwork. ➤ Absence of Schlemm's canal.		3. <u>Secondary infantile glaucoma:</u> <i>e.g.</i> ➤ Retinoblastoma. ➤ Intra-ocular inflammation. ➤ Trauma. ➤ Aniridia. ➤ Ectopia lentis. ➤ Sturge-Weber syndrome.	
Symptoms: ✎ <u>Early:</u> Photophobia, lacrimation and blepharo-spasm (nerve ending irritation due to corneal edema). ✎ <u>Late:</u> Large eye (ox eye) and corneal haziness.		Treatment: ☉ <u>Treatment of the cause and condition:</u> <i>varies acc. to corneal diameter and clarity:</i> ✎	
Signs: ✎ <u>Cornea:</u> Increase in diameter more than 12 mm, hazy and Haab's striae (fine horizontal curvilinear lines due to healed breaks in Descemet's membrane). ✎ <u>Sclera:</u> Stretched, thin so the uvea shines through it giving blue appearance. ✎ <u>Anterior chamber:</u> deep due to enlargement of the globe and displacement of the lens posteriorly. ✎ <u>Angle of the anterior chamber:</u> <i>viewed with Gonioscopy showing possible anomalies.</i> ✎ <u>Optic nerve:</u> glaucomatous cupping and atrophy (in late stages).		1. <u>Goniotomy:</u> ➤ Indication: corneal diameter is 13 mm or less with clear cornea. ➤ Principle: to incise the mesodermal membrane along 1/3 circumference of the angle	
		2. <u>Trabeculotomy:</u> ➤ Indication: corneal diameter is 13 mm or less with hazy cornea (i.e. anterior chamber angle is not well seen). ➤ Principle: to incise Schlemm's canal towards the anterior chamber angle.	
		2. <u>Trabeculectomy:</u> <i>done alone or with adjuvant Mitomycin-C</i> ➤ Indication: corneal diameter is more than 13 mm with hazy cornea ➤ Principle: external Fistulizing operation communicating AC the sub-tenon or sub-conjunctival space.	
		3. <u>Peripheral iridectomy (Incomplete or button hole iridectomy):</u> ✎ <u>Means:</u> excision of a portion of the iris near its root (here the pupil remains round). ✎ <u>Indications:</u> usually performed as a part of other cataract or glaucoma operations / or in the treatment of PCAG (In prodromal stage, in cases controlled medically before PAS occur and prophylactic against acute attack in the other eye).	
		4. <u>Visual iridectomy (Optical iridectomy):</u> ✎ <u>Means:</u> excision of small portion of the iris near the pupillary not reaching the ciliary border (Its site of choice is down and in to assist in convergence. It must be as small as possible to minimize dazzling). ✎ <u>Indications:</u> central localized opacities of the cornea and lens provided the visual acuity improves after dilatation (i.e. after positive mydriatic test). ☉ <u>Miscellaneous indications of iridectomy:</u> Iris prolapse (post-traumatic or post-operative), Iris FB and Iris Tumors (not reaching the ciliary border).	
Iridectomy			
Definition: ✎ Removal of a portion of the iris.			
Types and Indications: 1. <u>Key-hole iridectomy (Complete or sector iridectomy):</u> ✎ <u>Means:</u> excision of a part of the iris from the pupil to the ciliary border in one snip. ✎ <u>Indications:</u> usually performed as a part of other cataract or glaucoma operations.			
2. <u>Wide basal iridectomy (Basal iridectomy or broad basal):</u> ✎ <u>Means:</u> excision of a wide portion of the iris in two snips - in between, the iris is torn from its root. ✎ <u>Indications:</u> the classic treatment for acute congestive glaucoma which can't be controlled medically in 24 hours (i.e. substitute for SST).			

Retina			
	Central retinal artery occlusion	Central retinal vein occlusion	
		Non-ischemic	Ischemic type
Definition	- Occlusion of the central retinal artery (branch from the ophthalmic artery which is branch from the internal carotid artery) ⇒ irreversible ischemic retinal infarction. - Site of occlusion: at the level of lamina cribrosa.	Occlusion of the central retinal vein (which ends to the ophthalmic veins then the cavernous sinus).	
Etiology	- Thrombosis: following atherosclerosis in diabetic and hypertensive patients. - Embolism: either: - Detached thrombus e.g. in MI and SBE (main cause). - Air embolism. - Fat embolism e.g. in acute hemorrhagic pancreatitis. - Very high IOP: e.g. during RD surgery. - Spasm of the CRA: e.g. in Hypertensive encephalopathy and Migraine. - Arteritis: e.g. in Giant cell arteritis and Poly-arteritis nodosa.	- Thrombosis: following atherosclerosis in diabetic and hypertensive patients. - Blood disease: e.g. leukemia, polycythemia, sickle-cell disease. - High IOP. - Peri-phlebitis.	
Symptoms	Sudden unilateral painless <u>loss</u> of vision.	Sudden unilateral painless <u>moderate decrease</u> of vision (noticed more in early hours of the morning as during sleep, the circulation is sluggish).	
Signs	- V.A: severe impairment up to no PL. - External appearance of the eye: normal. - Pupil: dilated with loss of the direct light reflex and preservation of the consensual (RAPD). - Ophthalmoscopy (Fundus examination): - Vessels: narrowed (attenuated) arteries and arterioles with dark (due to stasis) segmented blood column. - Retina proper: milky white due to coagulative necrosis of the inner retinal layers and cloudy swelling of ganglion cells ⇒ extensive ischemic edema. - Macula: cherry red spot as the fovea receives its supply from the intact chorio-capillaries ⇒ retain its red color against the rest milky white retina. - Optic disc: consecutive optic atrophy (the end stage).	- V.A: moderate impairment. - External appearance of the eye: normal. - Pupil: sluggish direct light reflex and preservation of the consensual (RAPD). - Ophthalmoscopy (Fundus examination): - Vessels: tortuosity and dilatation of all branches of the central retinal vein. - Retina proper: hard exudates and hemorrhages, flame-shaped or punctate (dot-and-blot), are scattered in all quadrants. - Macula: edema (leakage edema – focal or diffuse). - Optic disc: edema. - Cotton wall spots (soft exudates): indicating capillary occlusion (micro-infarctions) – only in ischemic type. - N.B. All signs are milder in non-ischemic type with no cotton wall spots.	
Fate (Comp.)	- Irreversible loss of vision due to irreversible death of the inner half of the retina (supplied by CRA) in in 5-10 minutes. - The central vision may be preserved in 15% of the population who have additional artery supplying the macula area (cilio-retinal artery, arising from the posterior ciliary arteries). - After few weeks, The color of the fundus returns normal as macrophages engulf dead ganglion cells. Optic disc shows consecutive optic atrophy.	- Neovascularization: - Site: at the disc (NVDs) and elsewhere (NVEs) then at the iris (rubeosis iridis) and the angle of AC. - Sequelae: vitreous hemorrhage and Neo-vascular (100-day) glaucoma . - N.B.₁ Complications usually occur with the ischemic type. - N.B.₂ The neo-vascular glaucoma due to CRVO is called 100-day glaucoma as it usually occurs 100 days after the onset of the vein occlusion	
Investigations		- Fluorescein angiography: - Timing: After resolution of hemorrhages. - Aim: to detect ischemia, macular edema and neovascularization	

Treatment	✎ Cause: ☁ <u>Thrombosis:</u> Intra-venous Streptokinase (in first 24hours). ☁ <u>Raised IOP:</u> Ocular massage while the patient is lying flat – Anterior chamber paracentesis – Intra-venous Acetazolamide (500 mg). ☁ <u>Search for the underlying cause:</u> as it may be due to a life-threatening cardiovascular disease. ✎ <u>Condition:</u> inhalation of carbogen mixture (5% carbon dioxide and 95% oxygen) aiming to dilate obstructed CRA. ☠ <u>N.B.</u> The prognosis is very bad. However, all patients should be treated immediately as an emergency.	✎ Cause: ☁ <u>Thrombosis:</u> Antiplatelet medication like aspirin (75 mg / day). ☁ <u>Search for the underlying cause and control it:</u> as DM and Hypertension. ✎ Condition: <i>treatment of macular edema</i> ☁ <u>Laser treatment:</u> focal, grid or modified grid treatment. ☁ <u>Intra-vitreous injections:</u> of TAA or anti VEGF. ✎ Complications: ☁ <u>Argon laser photocoagulation:</u> used after hemorrhage resolve in ischemic cases and in cases with neo-vascularization up to neo-vascular glaucoma. ☁ <u>↓ IOP:</u> in cases with glaucoma by Insertion of glaucoma (Ahmed) valve, Molteno tube or doing cyclo-destructive operation (e.g. cyclo-cryotherapy).
	Branch retinal artery occlusion	Branch retinal vein occlusion
Main features	✎ Occlusion of one of the branches of the CRA (usually by emboli) ✎ Permanent visual field defect occurs corresponding to the occluded branch. ✎ <u>By ophthalmoscopy:</u> The occluded branch is narrowed (attenuated), with milky white area of infarction (necrosis) along its course.	✎ Occlusion of one of the branches (tributaries) of the central retinal vein. ✎ If the occluded branch is more peripheral ⇒ vision is not impaired. ✎ If the occluded branch is central ⇒ vision is impaired (due to macular edema). ✎ <u>By ophthalmoscopy:</u> The occluded branch is dilated and tortuous, with hard exudates, hemorrhages and edema along its course.
Retinitis pigmentosa		Complications:
Definition and Etiology:		✎ Complicated cataract. ✎ Consecutive optic atrophy. ✎ Macular affection (very late) ⇒ loss of visual acuity.
✎ Group of hereditary disorders characterized by progressive loss of photoreceptors (rod-cone dystrophy) and RPE function, resulting in retinal degeneration. ✎ The affection starts at the equator then spreads anteriorly then posteriorly. ✎ Definite cause is unknown, Inheritance plays a role (may be Autosomal dominant, Autosomal recessive or X-linked).		Prognosis:
Symptoms:		✎ Very poor.
✎ Asymptomatic ✎ Night blindness (defective dark adaptations). ✎ Progressive loss of vision and appearance of visual field defects.		Syndromes associated with retinitis pigmentosa:
Signs:		✎ <u>Laurence-Moon-Biedl syndrome:</u> <i>Retinitis pigmentosa plus:</i>
✎ Fundus examination (by ophthalmoscope): <i>triad of:</i> ➤ Arteriolar attenuation. ➤ Spider-like pigmentations at the equator and periphery of the retina. ➤ Later, waxy disc pallor of the optic disc due to consecutive optic atrophy.		➤ Mental retardation. ➤ Hypo-genitalism.
✎ Visual field examination (by perimetry): ➤ Ring scotoma (double arcuate scotoma) at the area 50 degree.		➤ Polydactyly. ➤ Obesity.
Investigations:		✎ <u>Refsum's disease:</u> <i>Retinitis pigmentosa plus:</i>
✎ Electro-retino-gram (ERG:) markedly diminished especially scotopic ERG.		➤ Deafness. ➤ Ataxia.
		➤ Polyneuropathy. ➤ Electro-oculogram (EOG) changes.
		Age-related macular degeneration
		Definition
		✎ Visual loss due to atrophy of the RPE in patients above the age of 50 years.
		Types: 2 types (dry and wet).
		☠ <u>N.B.</u> Wet ARMD is due to abnormal new vessels growing from the choroid under the retina resulting in macular edema with severe loss of vision.

	Diabetic retinopathy	Hypertensive retinopathies																																																					
Definition & Etiology	🗑 Bilateral affection of the retinal micro-vessels (capillaries, arterioles and venules) due to DM - (i.e. bilateral retinal micro-angiopathy). 🗑 Risk factors: ☁ Duration of DM: main RF (50% of DM patients ⇒ DR in 10 y). ☁ Metabolic control: good control delay not prevent DR. ☁ Miscellaneous factors: pregnancy, hypertension, RF, etc. ⇒ aggravate DR.	🗑 Bilateral affection of the retinal vessels due to hypertension (either benign, malignant due to RF of hypertensive toxemia of pregnancy).																																																					
	🗑 Micro-vascular leakage: ☁ Cause: breakdown of blood retinal barrier. ☁ Effects: micro-aneurysms, retinal hemorrhage, edema and hard exudate. 🗑 Micro-vascular occlusion: ☁ Cause: Anatomical (↑ed stickiness and aggregation of platelets) or functional (↓ed RBCs deformability). ☁ Effects: cotton -wall spots, IRMAs and neo-vascularization.	🗑 Vascular-constriction (of arterioles): ☁ Cause: vascular sclerosis or spasm. ☁ Effects: ischemic cotton -wall spots. 🗑 A-V crossing changes: ☁ Cause: vascular sclerosis. 🗑 Vascular leakage: ☁ Cause: breakdown of blood retinal barrier. ☁ Effects: retinal hemorrhage, edema and hard exudate ⇒ exudative RD.																																																					
Clinical types	1. Background Diabetic Retinopathy (BDR): ☁ Symptoms: usually asymptomatic. ☁ Signs (by ophthalmoscopy): micro-aneurysms, retinal hemorrhage (dot and blot), edema and hard exudate. 2. Pre-Proliferative Diabetic Retinopathy (PPDR): ☁ Symptoms: gradual diminution of vision. ☁ Signs (by ophthalmoscopy): as BDR + cotton -wall spots (may progress to extensive ischemic areas), IRMAs and venous changes (beading and looping). 3. Proliferative diabetic retinopathy (PDR) = Complications: ☁ Symptoms: symptoms of each complication (e.g. floaters). ☁ Signs (by ophthalmoscopy): Neo-vascularization (write as CRVO), fibrosis and opaque membranes around the new vessels ⇒ vitreous and tractional RD. 🗑 Diabetic maculopathy: ☁ Symptoms: acute ↓ of central vision (main cause of ↓ed vision in DR). ☁ Signs (by ophthalmoscopy): macular hemorrhage, edema or hard exudate. 💀 N.B. Macular edema in DR may be leakage (focal or diffuse), ischemic or mixed edema.	<table><tr><td></td><td>Benign hypertension</td><td>Malignant Hypertension and RF</td><td colspan="2">Toxemia of pregnancy</td></tr><tr><td>Pathogenesis</td><td>VC (sclerotic). A-V changes. Vascular leakage.</td><td>VC (sclerotic or spastic). Vascular leakage.</td><td colspan="2">VC (Spastic). Vascular leakage.</td></tr><tr><td>Symptoms</td><td colspan="4">Asymptomatic, blurring of vision with metamorphopsia (due to leakage) up to rapid painless ↓ of vision in exudative RD.</td></tr><tr><td>Hemorrhage</td><td colspan="4">Flame shaped (with other shapes in Malignant cases)</td></tr><tr><td>Edema</td><td>Mild (even absent)</td><td colspan="3">Marked ⇒ disc edema.</td></tr><tr><td>Hard exudate</td><td>Mild (deep round)</td><td colspan="3">Marked ⇒ Macular star (along Henle fibers).</td></tr><tr><td>Exudative RD</td><td>Usually absent</td><td colspan="3">Present (esp. in toxemia of pregnancy).</td></tr><tr><td>Cotton-wall S.</td><td>Usually absent</td><td colspan="3">Present.</td></tr><tr><td>Treatment</td><td>Control of HTN</td><td>Control of HTN and RF.</td><td colspan="2">Termination of pregnancy.</td></tr><tr><td>Prognosis</td><td>Good.</td><td>Death if untreated</td><td colspan="2">Good.</td></tr></table>					Benign hypertension	Malignant Hypertension and RF	Toxemia of pregnancy		Pathogenesis	VC (sclerotic). A-V changes. Vascular leakage.	VC (sclerotic or spastic). Vascular leakage.	VC (Spastic). Vascular leakage.		Symptoms	Asymptomatic, blurring of vision with metamorphopsia (due to leakage) up to rapid painless ↓ of vision in exudative RD.				Hemorrhage	Flame shaped (with other shapes in Malignant cases)				Edema	Mild (even absent)	Marked ⇒ disc edema.			Hard exudate	Mild (deep round)	Marked ⇒ Macular star (along Henle fibers).			Exudative RD	Usually absent	Present (esp. in toxemia of pregnancy).			Cotton-wall S.	Usually absent	Present.			Treatment	Control of HTN	Control of HTN and RF.	Termination of pregnancy.		Prognosis	Good.	Death if untreated	Good.	
		Benign hypertension	Malignant Hypertension and RF	Toxemia of pregnancy																																																			
Pathogenesis	VC (sclerotic). A-V changes. Vascular leakage.	VC (sclerotic or spastic). Vascular leakage.	VC (Spastic). Vascular leakage.																																																				
Symptoms	Asymptomatic, blurring of vision with metamorphopsia (due to leakage) up to rapid painless ↓ of vision in exudative RD.																																																						
Hemorrhage	Flame shaped (with other shapes in Malignant cases)																																																						
Edema	Mild (even absent)	Marked ⇒ disc edema.																																																					
Hard exudate	Mild (deep round)	Marked ⇒ Macular star (along Henle fibers).																																																					
Exudative RD	Usually absent	Present (esp. in toxemia of pregnancy).																																																					
Cotton-wall S.	Usually absent	Present.																																																					
Treatment	Control of HTN	Control of HTN and RF.	Termination of pregnancy.																																																				
Prognosis	Good.	Death if untreated	Good.																																																				
Investigations	🗑 Fluorescein angiography: ☁ Timing: After resolution of hemorrhages. ☁ Aim: to detect edema, ischemia and neovascularization.	Grading of hypertensive retinopathies: 🗑 Grade (1): 🗑 ☁ Vascular-constriction (of arterioles): mild generalized. 🗑 Grade (2): 🗑 ☁ Vascular-constriction (of arterioles): severe generalized & focal.																																																					
Treatment	🗑 Prophylaxis and Cause: Control DM and regular fundus examination: ☁ In DM without DR: examination yearly. ☁ In DM with BDR: every 9 months. ☁ In DM with PPDR or PDR: every 4 months. 🗑 Condition: treatment of macular edema (write as CRVO). 🗑 Complications: as CRVO + Vitrectomy. 💀 N.B. Rubeosis iridis with vitreous hemorrhage ⇒ vitrectomy and intraoperative pan-retinal photocoagulation (endo-laser).	☁ A-V crossing changes: venous deflection (Salus sign). 🗑 Grade (3): 🗑 ☁ Vascular-constriction (of arterioles): copper wiring. ☁ A-V crossing changes: venous deflection, distal banking and tapering. ☁ Vascular leakage: hemorrhage and hard exudate (also soft exudate). 🗑 Grade (4): 🗑 🗑 ☁ Vascular-constriction (of arterioles): silver wiring. ☁ A-V crossing changes: venous deflection, distal banking and tapering. ☁ Vascular leakage: as above + disc edema.																																																					

	Primary (Rhegmatogenous) Retinal Detachment	Secondary (Non-Rhegmatogenous) Retinal Detachment	
		Tractional	Exudative
Definition	✎ Separation of the inner sensory retina from the outer retinal pigment epithelium by sub-retinal fluid. ✎ Cause of separation: retinal break. ✎ Source of fluid: liquefied vitreous.	✎ Separation of the inner sensory retina from the outer RPE by sub-retinal fluid. ✎ Cause: sensory retina is pulled away by contracting fibrous tissues in vitreous. ✎ Source of fluid: Unknown.	✎ Site: retina is pushed off the choroid. ✎ Source of fluid: from choroid through damaged RPE.
	✎ Risk factors: ocular trauma, aphakia, family history and high myopia. ✎ Cause: Retinal break ☼ Definition: full-thickness defect in the sensory retina ☼ Shape, Site and Pathogenesis: ➤ Tears: horse-shoe (U-shaped) – temporal > nasal in upper fundus – produced by dynamic vitreoretinal traction – more dangerous. ➤ Holes: round or oval – upper > lower in temporal fundus – produced by chronic weakness and atrophy of the sensory retina – less dangerous. ➤ Retinal disinsertion: at the ora serrata.	✎ Proliferative DR (most common). ✎ Retinopathy of prematurity (ROP). ✎ Proliferative sickle cell retinopathy. ✎ Cyclitic membrane. ✎ Penetrating post. segment trauma.	✎ Choroidal tumors e.g. melanoma, hemangioma and metastases. ✎ Intraocular inflammation e.g. Harada's disease and posterior scleritis. ✎ Iatrogenic causes e.g. RD surgery and pan-retinal laser photocoagulation. ✎ Choroidal neo-vascularization ✎ Systemic causes e.g. malignant hypertension and toxemia of pregnancy.
Symptoms	✎ Flashes of light (Photopsia): ☼ Cause: traction at vitreoretinal adhesion sites. ☼ Cause of cessation: separation of the adhesion Or complete tearing away of a retinal piece (operculum) around the adhesion site. ✎ Visual field defect (Scotoma): ☼ Cause: spread of fluid behind the equator. ☼ Value: detection of the site of the retinal break. ✎ Central vision loss: ☼ Cause: spread of fluid to the fovea (foveal detachment). ✎ Floaters (musca volitantes): ☼ Definition: moving vitreous opacities perceived by the patient when they cast a shadow onto the retina. ☼ Cause: liquefied vitreous, tobacco dust and vitreous hemorrhage (2^{ry} to tearing of a peripheral retinal vessel ⇒ shower of minute dark red spots).	Complications of Primary (Rhegmatogenous) RD: ✎ Proliferative Vitreo-Retinopathy (PVR): (i.e. proliferation of retinal membranes) ☼ Site: Epi-retinal (on the inner retinal surface) or Sub-retinal (on the outer retinal surface). ☼ Signs: retinal folds and rigidity ⇒ restricted retinal mobility. ☼ Sequelae: postoperative contraction ⇒ failure in RD surgery. ✎ Others: e.g. irido-cyclitis, complicated cataract and vitreous hemorrhage.	
	✎ V.A: Depends on whether the fovea is involved (HM in total RD). ✎ IOP: ↓ed about 5 mmHg (due to absorption of SRF by the choroid). ✎ RR: grey. ✎ Slit-lamp examination: mild iritis and pigment cells (tobacco dust) in retro-lental vitreous. ✎ Ophthalmoscopy (Fundus examination): ☼ Retinal breaks: showing number, site, shape, size and extent (they appear as red discontinuities in the retinal surface). ☼ Sub-retinal fluid: which may extend up to the ora serrata and gives the retinal surface a wavy appearance. ☼ N.B. Retinal findings depend on the duration of RD and the presence of complications. ✎ B-scan Ultrasonography: to exclude 2^{ry} malignant exudative RD and to examine patients with opaque media (e.g. corneal opacities and cataract).	Treatment of Primary (Rhegmatogenous) RD: ✎ Prophylaxis: Retinal sealing ☼ Indications: ➤ Peripheral retinal lesions (risk factors). ➤ Flat retinal tears. ☼ Techniques: <i>surrounding the tear with:</i> ➤ 1 row of cryo-applications. ➤ 2-3 rows of argon laser photocoagulation (⇒ inflammatory chorio-retinal lesion ⇒ scarring ⇒ adhesion between sensory retina and RPE). ✎ Condition: Scleral Buckling (standard retinal surgery) – 3 steps: 1. Retinal sealing. 2. Scleral buckling: ➤ Technique: Creation of inward indentation of the sclera (buckle) at the site of retinal break(s) using an inert explant. ➤ Aim: closure of breaks and reduction of traction 3. With or without drainage of sub retinal fluid. ✎ Complications: Pars-plana vitrectomy ☼ Indications: ➤ PVR. ➤ Vitreous hemorrhage. ➤ Giant and Central breaks. ☼ Techniques: <i>removing the vitreous gel to gain access to the retina by 3 incisions:</i> ➤ Incision for introduction of infusion fluid ➤ Incision for illumination. ➤ Incision for vitreous cutter and aspiration.	

Neuro-Ophthalmology

Visual pathway	Light reflex pathway	Near reflex pathway
First Neuron: - Visual pathway starts from retinal photoreceptors (rods and cones). - Photoreceptors synapse with bipolar cells which then synapse with ganglion cells. Second Neuron: - Axons of the ganglion cells form the optic nerve which carries fibers from the temporal and nasal halves of the retina to optic chiasma. - In optic chiasma nasal fibers decussate to the contralateral optic tract whereas the temporal fibers pass in the ipsilateral optic tract. - Fibers leave the optic tract to end by synapsing in lateral geniculate body (LGB). Third Neuron: - Fibers from LGB pass through the optic radiation to synapse in calcarine area of the occipital cortex (visual cortex). - Visual cortex includes: - Primary visual area (area 17) – Striate cortex. - Visual association area (area 18) – Peri-striate cortex. - Para-striate cortex (area 19).	Stimulus: light (on one or both eyes). Receptors: retinal photoreceptors (rods and cones). Afferent: - The optic nerve which continue as the visual pathway till the post third of the optic tract where the fibers leave the tract. - Fibers enter the mid-brain and relay in the pre-tectal nucleus. - From the pre-tectal nucleus a new neuron (inter-calated neuron) delivers the impulses to Edinger-Westphal nucleus on both sides (i.e. fibers decussate again). Center: Edinger-Westphal nucleus (parasympathetic part of the third nerve nucleus). Efferent: along the 3rd nerve (oculomotor) to reach the ciliary ganglion ⇒ post ganglionic fibers enter the eye. Effector: the sphincter pupillae muscle Response: Bilateral miosis N.B. When light falls on one eye, its pupil becomes constricted (direct light reflex) and also the pupil of the other eye (consensual or crossed light reflex). N.B. Consensual or crossed light reflex is due to decussation of nerve fibers in optic chiasma and in mid-brain.	Stimulus: blurred image (from viewing near object). Receptors: retinal photoreceptors (rods and cones). Afferent: - The optic nerve which continue as the visual pathway till occipital cortex. - Fibers continue after that to the frontal cortex then the internal capsule delivering the impulses to the whole third nerve nucleus (including Edinger-Westphal nucleus). Center: third nerve nucleus (including Edinger-Westphal nucleus). Efferent: along the 3rd nerve (oculomotor) to reach the ciliary ganglion ⇒ post ganglionic fibers enter the eye. Effector: the sphincter pupillae muscle, the ciliary muscles, the medial recti muscles Response: Bilateral miosis, accommodation and convergence N.B. the 3 responses forming the near reflex are associated and that's called (synkinesis).

Field Changes due to Lesions in visual pathway

- Lesions in optic nerve:** monocular field defects on the same side with normal field on the other side.
- Lesions in optic chiasma:** bi-temporal hemianopia due to interruption of the nasal fibers of the optic nerve crossing at the chiasma (usually caused by pituitary tumors)
- Lesions in optic tract:** Contra-lateral homonymous hemianopia that respect the vertical meridian.
- Lesions in optic radiations:**
 - Temporal lobe lesions:** Contralateral homonymous superior Quadrantanopia with hemiparesis and dysphasia.
 - Parietal lobe lesions:** Contralateral homonymous inferior Quadrantanopia with agnosia.
- Lesions in occipital cortex:** contra-lateral homonymous hemianopia with macular sparing which is explained by:
 - Dual blood supply of the macular area (from the middle and the posterior cerebral arteries).
 - Bilateral representation of the macular fibers.
 - Large area of macular representation in the occipital cortex.

Causes of Miosis (Pupillary constriction)	Causes of Mydriasis (Pupillary dilatation)	Nystagmus												
1. Physiological causes: e.g. ✗ Light reflex and accommodation reflex. ✗ Third stage of general anesthesia. ✗ During sleep. ✗ Senile miosis. 2. Drugs: e.g. ✗ Local miotics e.g. Pilocarpine, Eserine, Di-isopropyl-fluoro-phosphate). ✗ Opium, morphine and parathion poisoning.. 3. Local Causes (ocular diseases): e.g. ✗ Hypermetropia ✗ Acute Irido-cyclitis. ✗ Traumatic miosis. ✗ Puncture of anterior chamber.. 4. Nervous causes: e.g. ✗ Homer's syndrome (ptosis, miosis, enophthalmos and Anhydrosis). ✗ Argyll Robertson's pupil. ✗ Pontine hemorrhage. ✗ Irritative stage of Hutchinson's Pupil.	1. Physiological causes: e.g. ✗ Withdrawal of light. ✗ Second and fourth stage of general anesthesia. ✗ Excitement, fear and anger. 2. Drugs: e.g. ✗ Local mydriatics e.g. Atropine, Homatropine, Hyoscine, Cocaine, Tropicamide and Cyclopentolate. ✗ Datura poisoning. 3. Local Causes (ocular diseases): e.g. ✗ High myopia. ✗ Acute congestive glaucoma. ✗ Traumatic mydriasis. ✗ Optic atrophy and retinal degeneration. ✗ Central retinal artery occlusion. 4. Nervous causes: e.g. ✗ All causes of coma except pontine hemorrhage, morphine and parathion poisoning. ✗ 3rd nerve (Oculomotor) paralysis. ✗ Paralytic stage of Hutchinson's pupil.	Definition: ✗ Involuntary oscillatory rhythmical movement of the eyes (usually bilateral and the movement is conjugate, coordinated and of equal intensity). Causes: 1. Ocular nystagmus:: e.g. ✗ Nystagmus due to defective central vision. ✗ Nystagmus due to blindness. ✗ Congenital idiopathic nystagmus. ✗ Spasmus Nutans. ✗ Miners disease. 2. Vestibular nystagmus: due to lesions of: ✗ labyrinth. ✗ Vestibular nerve 3. Central nystagmus: due to lesions of: ✗ Brains stem. ✗ Cerebellum. ✗ Spinal cord. Clinical forms: 1. Jerky nystagmus: has 2 phases: ✗ Slow movement in one direction. ✗ Quick correcting jerk in the other direction (direction of nystagmus). 2. Pendular nystagmus: both phases are of equal speed (usually of ocular origin). 3. Rotatory nystagmus: oscillatory movements occur around the visual axis. Clinical forms: ✗ Treatment of the cause. ✗ Optical treatment: correction of any error. ✗ Medical treatment: anti parkinsonian drugs. ✗ Surgical treatment: tree tenotomy of muscles												
Additional NBs: ✗ Functions of the pupil: ➤ Regulating the amount of light entering the eye. ➤ Cutting peripheral rays ⇒ ↓ spherical and chromatic aberrations of the eye. ➤ Increasing the depth of focus (which is the distance along which an object can be moved without becoming blurred for a known amount of accommodation, the smaller the pupil, the greater the depth of focus). ✗ Argyll Robertson Pupil: ➤ Definition: a small irregular pupil (mostly bilateral) which does not react to light but reacts to accommodation (i.e. light near dissociation) due to lesion in the intercalated neuron. This pupil dilates poorly in the dark even with atropine. ➤ Common causes: Neuro-syphilis, TD and general paralysis of insane GPI. ➤ Less common causes: DM, encephalitis and cerebral tumors. ✗ Hutchinson's pupil: caused by traumatic subdural hemorrhage ⇒ coma with following pupil changes: <table border="1"> <thead> <tr> <th></th><th>Ipsilateral pupil</th><th>Contralateral pupil</th></tr> </thead> <tbody> <tr> <td>Early</td><td>Constricted.</td><td>Normal reactive.</td></tr> <tr> <td>Advanced</td><td>Dilated not active.</td><td>Constricted.</td></tr> <tr> <td>More advanced</td><td>Dilated not active.</td><td>Dilated not active.</td></tr> </tbody> </table>				Ipsilateral pupil	Contralateral pupil	Early	Constricted.	Normal reactive.	Advanced	Dilated not active.	Constricted.	More advanced	Dilated not active.	Dilated not active.
	Ipsilateral pupil	Contralateral pupil												
Early	Constricted.	Normal reactive.												
Advanced	Dilated not active.	Constricted.												
More advanced	Dilated not active.	Dilated not active.												

	Papilledema	Optic neuritis			Optic atrophy
		Papillitis	Acute retro-bulbar	Chronic retro-bulbar	
Definition	🗑 Non-inflammatory passive edema of the optic nerve head (optic disc).	🗑 Inflammation of the optic nerve head (optic disc).	🗑 Acute Inflammation of the optic nerve behind the eyeball.	🗑 Chronic toxic Inflammation of the optic nerve behind the eyeball.	🗑 Degeneration of the axons of the ganglion cells (which forms the optic nerve).
Etiology	🗑 ↑ed ICP (main cause): due to ☁ Cerebral tumors. ☁ Subarachnoid hemorrhage ☁ Meningitis. ☁ Pseudo-tumor cerebri. 🗑 Other causes of swollen disc:	🗑 Demyelinating diseases: e.g. ☁ Disseminated sclerosis (DS). 🗑 Metabolic disorders and malnutrition e.g. ☁ DM. ☁ Vitamin B deficiency. ☁ Anemia. 🗑 General inflammations of nerves e.g. ☁ Neuro-myelitis Optica. ☁ Herpes-zoster (HZ). ☁ Neuro-Syphilis.	🗑 Exogenous toxins: either ☁ Tobacco, Ethyl alcohol (main cause ⇒ classic C/P discussed later). ☁ Quinine (⇒ constriction of the peripheral visual field i.e. tubular vision with cherry red spot in fundus examination). ☁ Methyl alcohol (⇒ severe optic atrophy).	🗑 Primary optic atrophy: due to lesions affecting ON behind eyeball ☁ CNS diseases e.g. DS - TD ☁ Blunt trauma to the ON. ☁ Toxins e.g. Tobacco. ☁ Pressure on the ON by tumors, aneurysm, etc. ☁ Severe blood loss, cardiac arrest and cardiac surgery.	
	☁ Systemic: ➤ Malignant hypertension. ➤ Severe anemia. ☁ Local: (usually unilateral) ➤ CRVO. ➤ Uveitis. ➤ Papillitis. ➤ Orbital cellulitis – CST. ➤ Hypotony (e.g. due to corneal fistula and choroidal detachment). 💀 N.B. optic disc edema due to ↑ed ICP ⇒ papilledema. But optic disc edema due to other causes ⇒ swollen disc.	🗑 Local inflammations (near the optic nerve) e.g. ☁ Uveitis. ☁ Orbital inflammations (orbital cellulitis and CST). ☁ Sinusitis. ☁ Meningitis. 🗑 Intoxication by: ☁ Lead, arsenic or ergot. Ischemic optic neuropathy: Definition: ischemic papillitis resulting from infarction of anterior optic nerve fibers (due to occlusion of the two posterior ciliary arteries). Causes: the same as CRAO. Complications: consecutive optic atrophy.		🗑 Secondary optic atrophy: due to lesions affecting ON head (OD) ☁ Papilledema. ☁ Papillitis.. 🗑 Consecutive optic atrophy: due to chorio-retinal diseases ☁ CRAO. ☁ RP. ☁ RD. ☁ Chorio-retinal degeneration ☁ DR. ☁ Chorio-retinitis. ☁ Ischemic optic neuropathy. 🗑 Glaucomatous optic atrophy: due to non-treated glaucoma	
Symptoms	🗑 Symptoms of the cause: headache, vomiting and diplopia 🗑 Vision: transient blurring. 🗑 V.F: central scotoma. 💀 N.B. OD has loose structure ⇒ vision isn't affected early.	🗑 Symptoms of the cause. 🗑 Vision: rapid loss of central vision 🗑 V.F: central scotoma.	🗑 Pain: dull aching exaggerated by ocular motility.	🗑 Symptoms of the cause: 🗑 Vision: gradual ↓ (misty). 🗑 V.F: Centro-cecal scotoma. 🗑 Difficulty to do close work. 🗑 Difficulty to discriminate colors.	🗑 Symptoms of the cause: 🗑 Vision: gradual painless ↓ (mainly in primary type). 🗑 V.F: marked peripheral constriction or lost V.D (mainly in primary type).
Signs	🗑 Pupil: normal. 🗑 V.F: enlarged blind spot then central scotoma to blue (later). 🗑 Fundus examination: see later.	🗑 Pupil: Marcus-Gunn pupil (RAPD) (i.e. sluggish unsustained direct and preserved indirect reflex). 🗑 V.F: central scotoma to red and green. 🗑 Fundus examination: see later.	🗑 Fundus examination: free. 🗑 Tenderness: on the pressing globe back	🗑 Pupil: Marcus-Gunn pupil.. 🗑 V.F: marked peripheral constriction then lost V.F. 🗑 Fundus examination: see later.	

Fundus exam. (ophthalmo scope)	Fundus examination of Papilledema: usually bilateral (due to ↑ed ICP).					
	🔪 Early stage: ☁ Vessels: tortuosity, engorgement and dilatation of the veins.					
	☁ Optic disc: ➤ Margins: blurred with flame shaped hemorrhage. ➤ Disc itself: mild swelling by edema (cup is filled). ☁ Retina proper and Macula: free.					
	🔪 Late stage: ☁ Vessels: Marked tortuosity, engorgement and dilatation of the veins + Loss of normal venous pulsations.					
	☁ Optic disc: ➤ Margins: blurred with flame shaped hemorrhage and hard exudates ➤ Disc itself: elevation by edema up to 8-10 D (Champaign Cork appearance or mushroom shaped). ☁ Retina proper: flame shaped hemorrhage, edema and hard exudates. ➡ ☁ Macula: Macular fan.					
	Fundus examination of Papillitis: usually unilateral					
	☁ Vessels: tortuosity, engorgement and dilatation of the veins (less than papilledema) – narrowing of the arteries. ☁ Vitreous inflammatory cells.					
	☁ Optic disc: ➤ Margins: blurred with flame shaped hemorrhage and hard exudates. ➤ Disc itself: hyperemic with mild elevation by edema up to 3D.					
	Fundus examination of Optic atrophy = Types (DD) of optic atrophy: variable acc. to each type					
		<u>Primary optic atrophy</u>	<u>Secondary optic atrophy</u>	<u>Consecutive optic atrophy</u>		<u>Glaucomatous optic atrophy</u>
	<u>Cause</u>	DS, TD and blunt trauma	Papilledema and Papillitis	<u>CRAO</u>	<u>Retinitis pigmentosa</u>	Untreated glaucoma
	<u>Retina proper</u>	Normal	Central pigmentary (edema, exudate, etc.)	Transparent pigmentary (Milky white retina)	Bone corpuscle (spider like) pigmentations	Tigroid fundus (RNFL defects)
	<u>Vessels</u>	Normal	Congested veins - attenuated sheathed ar.	Attenuated (thread like) arteries and arterioles	Attenuated (marked) arteries and arterioles	Nasal shift of vessels – interrupted
	<u>OD margins</u>	Well defined	Ill-defined (blurred)	Well defined	Slightly ill-defined	Overhang edges
	<u>OD Cup</u>	Saucer shaped	Obliterated	Saucer shaped	Obliterated	Deep large
	<u>OD lamina cribrosa</u>	Well seen	Obscured	Well seen	Obscured	Well seen
	<u>OD color</u>	White (milky)	Grey (pale)	White	Waxy yellow	White (pale)
Comp.	🔪 Secondary optic atrophy.		🔪 Primary optic atrophy (some consider it secondary type).			
Treatment	🔪 Treatment of the cause. 🔪 Stop tobacco, alcohol and give vitamin B, vasodilators esp. in chronic toxic optic neuritis.					

	Intra-ocular tumors	
	Malignant Melanoma of the choroid	Retinoblastoma
Origin	Melanocytes.	Primitive retinoblasts.
Incidence	- Most common primary intraocular tumor in adults. - Unilateral – Uni-centric tumor. - Rare in black races – No role for hereditary.	- Most common (1 in 20,000 live births) primary intraocular tumor in children (before 3 years when retinoblasts disappear) - Bilateral in 20% of cases – multi-centric tumor. - Caused by mutation in a gene on chromosome 13 – hereditary plays some role (positive family history is present in 6% of cases).
Clinical stages	<u>Quiescent stage: according to the site:</u> - if the tumor is peripheral: the patient is asymptomatic and tumor may be accidentally discovered during routine examination. - if the tumor is central: the patient complains of decreased vision due to macular affection. - N.B. Later on: the patient complains of decreased vision due to malignant RD.	
	<u>Stage of secondary glaucoma: due to:</u> - The associated intra ocular inflammation. - Obstruction of the trabecular pores by the malignant cells <u>Stage of extra-ocular extension</u> through sclera ⇒ orbit ⇒ proptosis. <u>Stage of distant metastases</u> ⇒ lung, bone, liver and brain (⇒ death especially with MM).	
	<u>Obstruction of the epi-scleral veins by the growing tumor.</u>	
	<u>Fundus examination: by ophthalmoscope aiming to:</u> - Early diagnosis (many cases may be accidentally discovered in quiescent during routine examination).	
	Documentation and follow up	
Investigations	N.B.₁ The tumor appears as elevated, brown oval shaped mass which may be mottled with dark brown or black pigments.	- N.B.₁ The tumor appears as grayish white mass with areas of calcification. - N.B.₂ The examination should be bilateral.
	- Ultrasonography: aiming to: - Accurate detection of tumor size and height.	Ultrasonography.
	Differentiation from other tumors e.g. choroidal nevus and hemangioma	- X-ray. - CT scan showing tumor calcifications.
Treatment	<u>Quiescent stage:</u> - Small peripheral: local resection (choroidectomy). - Small central and medium: radiotherapy (Radioactive plaques of gamma irradiation are fixed to the globe). - large: trans-pupillary thermotherapy	
	N.B. TTT is recent technique applying heat to the tumor tissue after pupillary dilatation using a diode laser ⇒ cellular destruction and inhibition of DNA synthesis ⇒ ↓ed size).	
	<u>Stage of secondary glaucoma:</u> enucleation (with excision of a long stump of the optic nerve) preceded by external beam irradiation to lessen the risk of metastases. <u>Stage of extra-ocular extension:</u> orbital exenteration <u>Stage of distant metastases:</u> palliative (symptomatic) chemotherapy.	
	<u>Quiescent stage:</u> - Small: photocoagulation (by diode laser). - Medium: radiotherapy. - large: trans-pupillary thermotherapy or trans-scleral cryotherapy.	

Strabismus

Ocular movements	Extra-Ocular muscles
Unocular movements (Ductions): ◎ Definition: movements of each eye by the 6 extra-ocular muscles around the 3 axes of the eye (vertical, horizontal, anteroposterior). ◎ They include: ✌ ✌ ✌ 1. Adduction: inward movement along the vertical axis. 2. Abduction: outward movement along the vertical axis. 3. Elevation (Supraduction): upward movement along the horizontal axis. 4. Depression (Infraduction): downward movement along the horizontal axis. 5. Intorsion (Incycloduction): rotatory movement along the anteroposterior axis in which the superior pole of the cornea (12 O'clock point) moves medially. 6. Extorsion (Excycloduction): rotatory movement along the anteroposterior axis in which the superior pole of the cornea (12 O'clock point) moves laterally. ◎ Agonist muscle: primary muscle moving the eye in a certain direction. ◎ Antagonist muscle: muscle moving the eye in the opposite direction. ◎ Sherrington's law: Increased innervation of a muscle is automatically associated with reciprocal decreased innervation of its antagonist.	Origin: ✎ Four recti and superior oblique: originate from the annulus of Zinn (common tendinous fibrous ring around the optic foramen i.e. at the orbital apex). ✎ Inferior oblique: originate from the orbital floor lateral to the NLD opening. Insertion: ✎ Four recti: inserted in the sclera in front of the equator at variable distances from the limbus (they pass forward to reach their insertion). ✎ Superior oblique: inserted in the upper posterolateral part of eye (it passes forwards and medially to reach the trochlea then bends backward and laterally to reach its insertion) ✎ Inferior oblique: inserted in the lower posterolateral part of the eye 5 mm from optic nerve almost over the macula (it passes backwards and laterally to reach its insertion). Action: 1. Superior rectus: ✎ Primary action: elevation. ✎ Subsidiary: adduction and intorsion. ✎ Cardinal: elevation when the eye is abducted (outward). 2. Inferior rectus: ✎ Primary action: depression. ✎ Subsidiary: adduction and extorsion. ✎ Cardinal: depression when the eye is abducted (outward). 3. Medial rectus: ✎ Primary action: adduction. ✎ Subsidiary: none. ✎ Cardinal: adduction only. 4. Lateral rectus: ✎ Primary action: abduction. ✎ Subsidiary: none. ✎ Cardinal: abduction only. 5. Superior oblique: ✎ Primary action: intorsion. ✎ Subsidiary: depression and abduction. ✎ Cardinal: depression when the eye is adducted (inward). 6. Inferior oblique: ✎ Primary action: extorsion. ✎ Subsidiary: elevation and abduction. ✎ Cardinal: elevation when the eye is adducted (inward). ☠ N.B.1 Recti muscles are adductors (except LR) while oblique muscles are abductors. ☠ N.B.2 Superior muscles are intortors while inferior muscles are extortors. ☠ N.B.3 Muscle action is maximum when the muscle axis is parallel to the visual axis (so e.g. superior rectus maximally elevates the eye when it's abducted) and vice versa. Nerve supply: ✎ All muscles except LR and SO: supplied by the 3 rd cranial nerve (oculomotor). ✎ Lateral rectus: supplied by the 6 th cranial nerve (abducent). ✎ Superior oblique: supplied by the 4 th cranial nerve (trochlear).
Binocular movements (Versions and Vergences): 📖 Versions (Conjugate movements): ◎ Definition: simultaneous symmetrical movements of both eyes in same direction (by yoke muscles using Herring's law of equal innervation). ◎ Examples: 1. Dextroversion: movement of both eyes to the right (by simultaneous contraction of right lateral rectus and left medial rectus). 2. Levoversion: movement of both eyes to the left (by simultaneous contraction of left lateral rectus and right medial rectus). ◎ Yoke muscles (Contralateral synergists): pair of muscles (one from each eye) which contract simultaneously during version movements. ◎ Herring's law: yoke muscles have simultaneous symmetrical innervation flows to move both eyes in certain same direction. 📖 Vergences: ◎ Definition: simultaneous symmetrical movements of both eyes in opposite direction. ◎ Examples: 1. Convergence: inward movement of both eyes (to look to near objects). 2. Divergence: outward movement of both eyes (to look to far objects).	

Position (Directions) of gaze:

⊙ **Cardinal directions:** directions achieved by only one muscle 🙌🙌🙌

1. Upwards and outwards: achieved by superior rectus.

2. Downwards and outwards: achieved by inferior rectus

3. Inwards: achieved by medial rectus.

4. Outwards: achieved by lateral rectus.

6. Downwards and outwards: achieved by superior oblique.

5. Upwards and inwards: achieved by inferior oblique.

⊙ **Non-cardinal direction:** directions achieved by more than one muscle 🙌

1. Upwards (pure elevation): achieved by superior rectus and inferior oblique.

2. Downwards (pure depression): achieved by inferior rectus and superior oblique.

⊙ **Primary direction:** direction achieved when the two eyes are directed straight ahead and all extra-ocular muscle are relaxed. .

⚠ N.B.₁ Secondary direction are pure upwards, downwards, inwards and outwards.

⚠ N.B.₂ Tertiary direction are upwards with outwards, upwards with inwards, downwards with outwards and downwards with inwards.

Ocular motility test:

✎ Examine each eye for movements in the six cardinal directions of gaze (to detect any limitation of movements and know the responsible muscle).

✎ Examine the movements of the two eyes simultaneously in the six cardinal directions of gaze (to detect any limitation in conjugate eye movements).

Some basics about strabismus**Definitions you should know:**

✎ Nodal point: the point just anterior to the posterior lens capsule through which light rays undergo no refraction and must reach the macula.

✎ Optic axis: the line between the corneal center, nodal point and the retinal center.

✎ Visual axis: the line between the macula, nodal point and the object of regard.

✎ Muscle axis: the line between the origin and the insertion of the muscle.

✎ Orthophoria: the perfect equilibrium of the oculomotor apparatus.

✎ Strabismus (squint): the condition in which the visual axes of both eyes are not directed to the same object of regard.

✎ Angle of squint: the angle between the 2 visual axes of the 2 eyes.

✎ Primary angle of squint: the angle of squint when the patient is squinting with the diseased eye (fixing with the normal eye).

✎ Secondary angle of squint: angle of squint when the patient is squinting with covered normal eye (forced to fix with the diseased eye).

How to measure angle of squint?

1. Corneal reflection method:

✎ Principle :

➤ The patient is asked to look at a light source at a distance of 50 cm.

➤ We indicate the angle from the position of the light reflection on the cornea of the squinting eye.

✎ Interpretation: 🙌

➤ If light reflection is at the center of the pupil $\Rightarrow$ 0 degree (No squint).

➤ If it's at the edge of a normal sized pupil $\Rightarrow$ 15 degree.

➤ If it's midway between the edge of the pupil and the limbus $\Rightarrow$ 30 degree

➤ If it's at the limbus $\Rightarrow$ 45 degree.

➤ If it's outside the limbus $\Rightarrow$ more than 45 degree (add 7 degree for each 1mm)

2. Synoptophore.

Binocular single vision**Definition:**

✎ It's the ability of the brain to see single image of one object by using the two eyes.

Prerequisites: The object form 2 images (1 on each retina), the following items are necessary for the brain to collect them in 1 single image that we can normally see : 🙌

✎ The two images should be identical or nearly identical as regard size, shape, clarity, etc. (i.e. good vision in both eyes should be present e.g. no marked Anisometropia).

✎ The two identical images should fall on normal retinal correspondence (i.e. good extraocular muscle function in both eyes should be present e.g. no squint).

✎ Normal fusion center should be present (i.e. no ischemia, , no under development, etc.)

Grades: 🙌

⊙ **Grade I (Simultaneous perception):** the ability to see two objects at one time when they stimulate corresponding points on retina.

✎ Example: **Bird and cage**

➤ The right eye sees image of a bird. ➤ The left eye sees image of a cage.

➤ The two eyes can see a single image of the bird in its cage.

⊙ **Grade II (Fusion):** the ability fuse similar incomplete objects with control.

✎ Example: **two rabbits (with few differences)**

➤ The right eye sees image of a rabbit with tail and without ears.

➤ The left eye sees image of a rabbit without tail and with ears.

➤ The two eyes can see single image of a rabbit with tail and ears.

⊙ **Grade II (Stereopsis):** the ability to perceive depth when each eye is presented with slightly dissimilar image of the same object

Binocular double vision

Components (Types):

📖 **Confusion:** seeing two images of two different objects superimposed (confused) on each other as they stimulate corresponding retinal foveas.

💀 **N.B.** normally the two foveas should be stimulated by one not two objects.

🔍 **Cause:** squint ⇒ mal-direction of one visual axis ⇒ won't meet the other axis at the point of fixation but fixate to different point.

🔍 **Example:** **circle and triangle**

- The normal right eye sees (fixate to) a circle.
- The squinting left eye sees (fixate to) a triangle.
- The two eyes see a confused image of the circle on the triangle.

📖 **Diplopia:** seeing two images of one object as they stimulate non-corresponding retinal points.

💀 **N.B.** normally the one object should stimulate two corresponding retinal points.

🔍 **Cause:** squint ⇒ deviation of one eye while the other is straight ⇒ the seen object stimulates non-corresponding retinal points.

🔍 **Example:** **circle**

- The normal right eye sees the clear circle (by the fovea)
- The squinting left eye sees the blurred circle (by another retinal point as this eye is deviated).
- The two eyes see two circles (one is clear and the other is not clear).

Adaptive mechanisms to overcome double vision:

📖 **Sensory mechanisms:** ✌ ✌

1. Suppression:

- **Prerequisites:** children + concomitant squint.
- **Principle:** sub-conscious active inhibition of vision in squinting eye by visual cortex.

💀 **N.B.** suppression is temporary (during binocular vision) i.e. if the fixing eye is covered, the suppression stops immediately and the squinting eye takes up fixation.

2. Amblyopia:

- **Prerequisites:** children + unilateral concomitant squint.
- **Principle:** prolonged suppression the squinting eye ⇒ decreased vision in this eye without any organic lesion.

💀 **N.B.** In alternating squint, no prolonged suppression to one eye ⇒ no amblyopia.

3. Abnormal retinal correspondence (ARC):

- **Prerequisites:** children + unilateral concomitant squint.
- **Principle:** prolonged amblyopia in the squinting eye ⇒ stimulating extra-foveal point to correspond the normal one of the other eye (false fovea).

💀 **N.B.** ARC is temporary (during binocular vision).

4. Eccentric fixation:

- **Prerequisites:** children + unilateral concomitant squint.
- **Principle:** prolonged ARC ⇒ the new false fovea becomes permanent and the patient fixate with it even when the other eye is covered.

💀 **N.B.** Eccentric fixation is permanent i.e. if the normal eye is covered, the squinting doesn't move but continues to fixate by its new false fovea.

📖 **Sensory mechanisms:** ✌ ✌

1. Latent squint:

- **Prerequisites:** small angle of squint + good binocular function.
- **Principle:** fine adjustment of the extraocular muscle tone ⇒ straightening of the eye (making the deviation latent).

💀 **N.B.** 1 If the patient get fatigued or the binocular vision is disrupted (e.g. by covering the diseased eye) ⇒ the deviation in the covered eye becomes manifest.

2. Purposive squint (Blind spot syndrome):

- **Prerequisites:** larger angle of squint + good binocular function.
- **Principle:** alternation of the extraocular muscle tone ⇒ throw the unwanted image on the optic disc (Blind spot).

3. Compensatory head posture:

- **Prerequisites:** fairly large angle of squint + fully developed binocular function + paralytic squint (complete paralysis of one or more of EOMs).
- **Principle:** turning of the head in the same direction of the paralyzed muscle action (as this patient complains of diplopia at this direction).

➤ Components (Types / Variants):

- 🌀 Face turn (in horizontal recti paralysis).
- 🌀 Chin elevation or depression (in vertical recti paralysis).
- 🌀 Head tilt (in oblique muscle paralysis).

Assessment (Evaluation):

1. Synoptophore:

- 🔍 **Aim:** detect the actual grade of binocular single vision.
- 🔍 **How does it work?** It consists of two tubes through which the patient sees, at the end of each tube, a slide can be put to view separate images.
- 🔍 **Interpretation:** (refer to grades of binocular single vision).

2. Worth's 4 dots test:

- 🔍 **Aim:** give us an idea about presence of binocular single or double vision or even presence of sensory mechanisms trying to overcome it.
- 🔍 **How does it work?** See next page 😊

How does it work?

- We have 4 colored illuminated dots (1 red, 1 white and 2 green).
- The patient wears red-green goggles (red in front of the right eye and green in front of the left eye) at a distance of 6 meters and view the dots.
- Now, the right eye sees 2 red dots (the original red and white dots appear red while green is not seen as it appears black).
- While, left eye sees 3 green dots (the original green and white dots appear green while red is not seen as it appears black).
- The patient is asked about the number and color of the dots he sees binocularly.

Interpretation: 🖐️

- If the patients sees 4 dots (1 red, 2 green and 1 red to green):
 👁️ + normal eyes ⇒ normal person. 👁️ + manifest squint ⇒ ARC or eccentric fix.
- If the patients sees 5 dots (2 red and 3 green). ⇒ diplopia (each eye sees separately)
- If the patients sees 2 red dots only. ⇒ left suppression (left eye doesn't see).
- If the patients sees 3 green dots only. ⇒ right suppression (right eye doesn't see).

Latent strabismus (Heterophoria)**Definition:** *Type of squint in which* 🖐️

- ✎ The eye has tendency to deviate from the orthophoric position (straight head).
- ✎ The brain checks (corrects) this tendency sub-consciously to maintain binocular single vision (i.e. so the squint is latent).
- ✎ This latent squint becomes manifest when:
 - ☁ Patient is fatigued. ☁ Brain loses interest in binocular single vision.
 - ☁ Binocular vision is disrupted (e.g. by covering one eye ⇒ its deviation).

Etiology: *Extra-ocular muscle imbalance due to* 🖐️

- ⊙ Uncorrected errors of refraction: *e.g.*
 - ✎ Hypermetropia: as the patient uses excessive accommodation to see clearly ⇒ associated excessive convergence ⇒ latent convergent squint (esophoria)
 - ✎ Myopia: as the patient relaxes his accommodation ⇒ associated lack of convergence ⇒ latent divergent squint (exophoria).
- ⊙ Mild weakness (not paralysis) of one or more of extraocular muscles.

Types: ✎ Esophoria (tendency to deviate in), Exophoria, Hyperphoria and Hypophoria.**Clinical picture:** *Extra-ocular muscle imbalance due to* 🖐️

- ⊙ Compensated cases: asymptomatic (present in a large percentage of the population)
- ⊙ De-compensated cases:
 - Symptoms due to trial to maintain binocularity e.g. muscular asthenopia ☺
 - Symptoms due to failure to maintain binocularity e.g. intermittent squint and diplopia

Evaluation: 🖐️**1. Cover uncover test:**✎ Principle: abolishing binocular vision by covering one eye ⇒ its deviation.✎ Steps: 1. Ask the patient to fixate to an object e.g. your finger.2. Cover one eye by piece of paper then uncover it observing its position.✎ Interpretation:

➤ If the person is normal: the eye doesn't deviate under the cover.

➤ If latent squint is present: the eye deviates under the cover and corrects its position to take fixation again when the cover is removed.

2. Maddox rod test: (for far diagnosis of Heterophoria)✎ Principle: abolishing binocular vision by providing each eye with a different image using a Maddox rod.✎ Steps: 1. Ask the patient to fixate to a source of light at a distance of 6 meters.

2. Place the Maddox rod in front of one eye (while the other eye is free)

✎ Interpretation:

➤ If the person is normal: he sees the point of light (formed by the free eye) coinciding with the middle of the red line (formed by the eye covered by the rod).

➤ If latent squint is present: he sees the point of light (formed by the free eye) on either side of the red line (formed by the eye covered by the rod).

3. Maddox wing test: (for near diagnosis of Heterophoria)✎ Principle: abolishing binocular vision by providing each eye with a different image using a Maddox wing.✎ Interpretation:

➤ If the person is normal: he sees arrows point to zero on scales.

➤ If latent squint is present: he sees arrows point to another number on scales (this number is considered his angle of squint).

☠ N.B. 1 In Maddox rod test, angle of squint can be measured by finding the prism which makes light coincide with the red line while In Maddox wing, the patient can directly reads his angle.**Treatment:**⊙ Treatment of the Cause: by correction of errors of refraction.⊙ Treatment of the Condition: **Surgical correction**✎ Indications: 🖐️

- ☁ When symptoms are not relieved by glasses (very rare).
- ☁ Non-refractive weakness of muscles.

✎ Principle: is to strengthen the underacting muscle or weaken the overacting muscle.☠ N.B. Compensated cases doesn't require treatment.

	Paralytic Strabismus	Concomitant Strabismus
Definition	🌀 Type of manifest squint in which: 🖐️ 👁️ The eye is deviated and its movement is limited due to paralysis of one or more of extraocular muscles. 👁️ The angle of squint is variable in different directions of gaze and also variable depending on which eye is fixing and which eye is squinting (i.e. the 2^{ry} angle of squint is larger than the 1^{ry} angle).	🌀 Type of manifest squint in which: 🖐️ 👁️ The eye is deviated without limitation of movements or paralysis of any extraocular muscles. 👁️ The angle of squint is constant in different directions of gaze and also constant whatever which eye is fixing and which eye is squinting. (i.e. the 2^{ry} angle of squint is equal to the 1^{ry} angle)
Etiology	Extra-ocular muscle paralysis due to LMNL e.g. ⦿ Nuclear lesions: which may be: 👁️ <u>Congenital:</u> absence of the nucleus. 👁️ <u>Inflammatory:</u> encephalitis. 👁️ <u>Vascular:</u> hemorrhage, thrombosis or embolism. 👁️ <u>Neoplastic:</u> brain tumors. ⦿ Nerve lesions: which may be: 👁️ <u>Traumatic:</u> fracture base of the skull. 👁️ <u>Inflammatory:</u> neuritis e.g. diabetes and diphtheria (toxic neuritis). 👁️ <u>Vascular:</u> subarachnoid hemorrhage and cavernous sinus thrombosis. 👁️ <u>Neoplastic:</u> brain tumors (due to increased intracranial pressure). ⦿ Muscle lesions: which may be: 👁️ <u>Congenital:</u> mal-development of the muscle. 👁️ <u>Traumatic:</u> fracture orbital bones. 👁️ <u>Neuro-muscular:</u> myopathy and myasthenia gravis. 👁️ <u>Neoplastic:</u> orbital tumors. 💀 N.B. UMNL don't produce paralysis of individual muscle but paralysis of conjugate movements ⇒ the patient can't look to the right or left etc.	"EOM imbalance -with no binocular vision memory i.e. in child- due to: ⦿ Uncorrected errors of refraction: e.g. 👁️ <u>Hypermetropia:</u> as the child uses excessive accommodation to see clearly ⇒ associated excessive convergence ⇒ concomitant convergent squint (accommodative esotropia). 👁️ <u>Myopia:</u> as the child relaxes his accommodation ⇒ associated lack of convergence ⇒ concomitant divergent squint (accommodative exophoria). ⦿ Congenital motor weakness of one or more of extraocular muscles (congenital esotropia much more common than exotropia) ⦿ Sensory monocular decreased vision (secondary squint): e.g. 👁️ <u>Unilateral corneal opacity.</u> 👁️ <u>Unilateral lens opacity (congenital cataract).</u> 👁️ <u>Unilateral retinal detachment.</u> 👁️ <u>Unilateral intraocular tumor (retinoblastoma).</u> 💀 N.B. 1 These sensory obstacles interferes with developing binocular single vision ⇒ the eye suppress the diseased eye ⇒ squint. 💀 N.B. 3 Squint is usually convergent if occurred in early years of childhood. 💀 N.B. 4 Squint is usually divergent if occurred later on (resting position) ..
Diagnosis	Clinical picture and evaluation: 🖐️ 🖐️ 1. <u>Ocular deviation:</u> to the opposite direction of the paralyzed muscle action. (the paralyzed muscle loses its tone ⇒ the antagonist draws the eye towards it). 2. <u>Limitation of ocular movement:</u> in the same direction the paralyzed muscle action (diagnosed by ocular motility test). 🌀 How to asses ocular motility? 3. <u>Angle of squint:</u> changes in different directions of gaze (larger in the same direction the paralyzed muscle action) and also changes depending on which eye is fixing (the secondary angle is larger than the primary angle). 💀 N.B. secondary angle of squint is larger as when the paralyzed muscle tries to fixate ⇒ the brain sent excessive impulses to help it ⇒ the same excessive impulses also reach the normal yoke muscle in the other eye (Herring's law) ⇒ larger deviation. 🌀 Define angle of squint? Types? How to measure?	Sequelae: discussed before in details (see diplopia). 1. Suppression. 2. Amblyopia. 3. Abnormal retinal correspondence (ARC). 4. Eccentric fixation. Directions: 👁️ <u>Convergent squint:</u> the eye deviates inwards. (usually in early childhood). 👁️ <u>Divergent squint:</u> the eye deviates outwards (usually later on). 🌀 How to differentiate? 📖 Corneal reflection method: 👁️ <u>Principle:</u> noticing the direction of light reflection when it falls on the eye. 👁️ <u>Steps:</u> ask the patient to look at a light source at a distance of 50 cm and notice the corneal reflection of the light. 👁️ <u>Interpretation: if convergent::</u> light image deviates temporally (and vice versa).

Diagnosis	4. <u>Binocular diplopia</u>: maximum when the patient looks at an object situated in the same direction of the paralyzed muscle action. ✎ <u>N.B.</u> Binocular diplopia disappears when one eye is covered. ✎ <u>N.B.</u> The false image is the one seen by the squinting eye; it is blurred because it falls outside the macula. 🌀 Define diplopia? How to measure? 5. <u>Compensatory head posture</u>: abnormal head deviation to the same direction of the paralyzed muscle action (to avoid diplopia). 🌀 Enumerate Components of compensatory head posture? 6. <u>False projection</u>: wrong estimation of the sites of objects situated in the same direction of the paralyzed muscle action. Examples of some paralytic palsies:	Types: ✎ <u>Unilateral squint</u>: one eye is always squinting all the time, and the other eye is always fixing (so amblyopia occurs in the squinting eye). ✎ <u>Alternating squint</u>: each eye can maintain fixation, i.e. they alternate with each other (so, no amblyopia occurs).
	🌀 Right Abducent or sixth nerve palsy (lateral rectus palsy)	🌀 How to differentiate?
	✎ the right eye is deviated inwards (convergent squint). ✎ Limitation of movement when the patient moves his eye to the right. ✎ The angle of squint is large when the patient looks to the right side. ✎ The angle of squint is large when the right eye is fixing (left eye is now squinting). ✎ Diplopia is most marked when the patient looks to the right. ✎ Face turn to the right (compensatory head posture).	📖 Cover test: ✎ <u>Principle</u>: making both eye alternate the fixation. ✎ <u>Steps</u>: 1. Cover the apparent normal eye and ask the patient to look to your finger (now the squinting eye is forced to take fixation). 2. Remove the cover and notice the uncovered (fixing) eye. ✎ <u>Interpretation</u>: ☁ <u>If the squint is unilateral</u>: the covered previously normal eye returns to take fixation (the previously deviating eye will deviate again). ☁ <u>If latent squint is present</u>: the uncovered previously squinting eye keep its new fixing position (i.e. they alternate before and after test). 📖 Visual acuity: ✎ <u>Principle</u>: ☁ <u>If the squint is unilateral</u>: the squinting eye shows marked diminution of vision than the normal eye because of amblyopia. ☁ <u>If the squint is alternating</u>: vision is equal or nearly equal in both eyes as there is no amblyopia.
	🌀 Right Trochlear or fourth nerve palsy (superior oblique palsy)	Clinical picture and evaluation: 🖐 🖐 📖 History taking: 1. <u>Age of onset</u>: e.g. ✎ <u>Infantile congenital squint</u>: within the 1st 6 months (usually needs surgery). ✎ <u>Refractive squint</u>: later on (2-3 years in accommodative esotropia). 2. <u>Family history</u>: strabismus is frequently hereditary. 3. <u>Variability</u>: e.g. If the deviation is worse when the child is tired or ill ⇒ accommodative element is likely to be present. 4. <u>Intermittent or constant</u>: If intermittent ⇒ prognosis is better (as some binocular single vision is still present). 5. <u>Diplopia</u>: usually absent (as the child can compensate it through sensory mechanisms – sequelae).
	✎ The right eye is deviated upwards and slightly inwards with extortion ✎ Limitation of movement when the patients moves his eye down and in (much discomfort on going downstairs). ✎ The angle of squint is large when the patient looks down and in. ✎ The angle of squint is large when the right eye is fixing (left eye is now squinting). ✎ Diplopia is most marked when the patient looks on looking down and in. ✎ Chin depression with face turn to left and head tilt towards left shoulder (in the same direction of superior oblique action = down and in).	

Diagnosis	<i>Oculomotor or third nerve palsy</i> ✎ Ptosis. ✎ Dilatation of the pupil. ✎ Paralysis of accommodation. ✎ Deviation of the eye down (superior oblique) and out (lateral rectus). ✎ Limitation of movement in the same direction of action of the involved muscles. ✎ No diplopia (due to ptosis). Note the following differences: ✎ Ophthalmoplegia: paralysis of ocular muscles, <i>which may be:</i> ✎ Internal ophthalmoplegia: paralysis of intra-ocular muscles. ✎ External ophthalmoplegia: paralysis of extra-ocular muscles.. ✎ Total ophthalmoplegia: paralysis of intra-ocular and extra-ocular muscles ✎ Superior orbital fissure syndrome: <i>combination of:</i> ✎ Paralysis of ocular motor n. supply (3rd, 4th, 6th cranial nerves). ✎ Paralysis of ocular sensory n. supply (ophthalmic division of 5th cranial n.). ✎ Orbital apex syndrome: <i>combination of:</i> ✎ Paralysis of ocular motor n. supply (3rd, 4th, 6th cranial nerves). ✎ Paralysis of ocular sensory n. supply (ophthalmic division of 5th cranial n.). ✎ Paralysis of the optic nerve (optic atrophy)	📖 Clinical examination: 1. Prove that's concomitant squint (i.e. exclude paralytic squint): <i>by:</i> ✎ Testing the ocular motility. ✎ measurement of angle of squint. <i>How to asses ocular motility?</i> <i>Define angle of squint? Types? How to measure?</i> <i>describe angle of squint in concomitant squint?</i> ⚠ N.B. angle of squint is usually large in congenital squint. 2. Detect the type (unilateral – alternating): <i>define concomitant squint types? How to differentiate?</i> 3. Detect the direction (Convergent – Divergent): <i>define concomitant squint directions? How to differentiate?</i> 4. Detect the etiology: ✎ Exclude secondary squint (by careful examination of the eye both anterior and posterior segments). ✎ differentiate between primary congenital and accommodative esotropia (by examining the refraction of the eye). <i>enumerate causes of secondary squint?</i> 5. Detect the sequelae (state of binocular vision): <i>How to measure the binocular vision state?</i>
	Amblyopia Definition: Impaired vision in the absence of organic disease usually due to lack of continuous use of one or both foveas for vision. Causes (Types): 1. Strabismic amblyopia: to avoid diplopia, the brain suppresses the image of the squinting eye which over time 2. Anisometropic amblyopia: due to marked difference in refraction between the two eyes ⇒ The brain suppresses the blurred image of the eye having the higher error. 3. Visual deprivation amblyopia (amblyopia ex anopsia): due to opacities in the ocular media as unilateral congenital cataract or ptosis covering the papillary area. 4. Toxic amblyopia: due to chronic retro-bulbar neuritis. 5. Nystagmic amblyopia. Treatment: ☉ <u>Treatment of the cause:</u> ☉ <u>Treatment of the condition:</u> Occlusion (patching) of the preferred eye ✎ Aim: forcing the patient to use the amblyopic eye for vision. ✎ Timing: as early as possible (before age 9); otherwise occlusion will not improve V.A.	Pseudo-strabismus (False - Apparent squint) Definition: It is a condition in which there's false impression of ocular deviation but on examination eyes are orthophoric with normal binocular single vision. Causes (Types): ✎ <u>Apparent convergent squint:</u> ➤ Small inter-pupillary distance. ➤ Epicanthus: Prominent folds hiding part of the normally visible sclera. ✎ <u>Apparent divergent squint:</u> ➤ Large inter-pupillary distance. Diagnosis: 1. <u>Corneal light reflex test:</u> normally centered. 2. <u>Cover test:</u> no movements occur. Uses of Synoptophore ☉ <u>Diagnostic:</u> e.g. Measures of the angle of the squint, the angle alpha, the grade of binocular vision and Detects suppression. ☉ <u>Therapeutic:</u> e.g. Improves the grade of binocular vision and treats suppression